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Veterinary Remedies Under the New Food, Drug and Cosmetic Act*

By H. E. MOSKEY, *Washington, D. C.*

Food and Drug Administration, U. S. Department of Agriculture

VETERINARIANS, live-stock and poultry raisers, as well as manufacturers of veterinary remedies, have known for some time the activities of the Food and Drug Administration in the enforcement of the Food and Drugs Act of 1906. On June 25, 1938, the new Food, Drug and Cosmetic Act became a law.

TERMS DEFINED

The act defines the term "drug" to mean articles recognized in the official United States Pharmacopoeia, official Homeopathic Pharmacopoeia of the United States, or official National Formulary, or any supplement to any of them; articles intended for use in the diagnosis, cure, mitigation, treatment or prevention of disease in man or other animals; and articles (other than food) intended to affect the structure or any function of the body of man or other animals.

The new act also defines the term "device" to mean instruments, apparatus and contrivances, including their components, parts and accessories, intended for use in the diagnosis, cure, mitigation, treatment or prevention of disease in man or other animals; or to affect the structure or any function of the body of man or other animals. Therefore, all sections of the new act dealing with drugs and devices, with the exception of that section dealing with habit-forming drugs, are applicable to veterinary remedies.

Most of the provisions of the new act will not become effective until one year from the date of its passage. The provisions which are now in effect apply to new drugs and those drugs which are dangerous to health when used in the dosage or with the frequency or duration prescribed, recommended or suggested in the labeling thereof.

"NEW DRUG" PROVISION

Regulations for the enforcement of the "new drug" provision have already been approved. Under the regulations a drug may be a new drug by reason of (1) the newness for drug use of any substance which composes the drug, in whole or in part, whether it be an active substance or a menstruum, excipient, carrier, coating, or other component; (2) the newness for drug use of a combination of two or more substances, none of which is a new drug; (3)

*Presented at the Nineteenth Annual Veterinary Conference, University of Illinois, Urbana, Ill., October 5, 1938.

the newness for drug use of the proportions of the substances in a combination, even though the substances in certain other proportions do not constitute a new drug; or (4) the newness of the dosage, or methods or duration of administration or application, or other conditions of use of the drug prescribed, recommended or suggested in the labeling thereof, even though the drug is not a new drug when used in certain different dosage, or in different methods or duration of administration or application, or in conditions that are otherwise different.

These sections of the act dealing with new drugs require the manufacturer of a new drug to file an application with the Secretary of Agriculture; a full report of investigations which have been made to show whether or not such drug is safe for use; a full list of the articles used as components of such drug; a full statement of the composition of such drug; a full description of the methods used in, and the facilities and controls used for, the manufacture, processing and packing of such drug; such samples of such drug and of the articles used as components thereof as the Secretary may require; and specimens of the labeling proposed to be used for such drug.

New Drugs for Investigational Use

If the Secretary finds, before the date prescribed by the act for an application to become effective, that he has no cause to issue an order under the act refusing to permit the application to become effective, the Food and Drug Administration shall so notify the applicant in writing, and the application shall become effective on the date of the notification. However, an exemption is made in the case of a new drug if it is intended solely for investigational use by experts qualified by scientific training and experience to investigate the safety of drugs; if the labeling bears the statement, "For investigational use only"; if it is delivered only to such experts and is used by them solely for investigation; if such expert has adequate facilities for the investigation to be conducted by him, and if the person who introduced the drug in interstate commerce or delivered it for such introduction holds a signed statement from each expert to the effect that he will not use the drug otherwise than in the investigation to be conducted by him unless and until an application to the Secretary becomes effective with respect to such drug.

Labeling Must Be Accurate

While no regulations have as yet issued concerning those sections of the new act which do not go into effect until June 25, 1939, some of the requirements under these sections may be mentioned. Drugs and devices, when shipped in interstate commerce, must bear the name and place of business of the manufacturer, packer or distributor. The labeling of a drug must bear an accurate statement of the quantity of the contents, and the common name of the drug, and in the case of non-official drugs manufactured from two or more ingredients, the common or usual name of each active ingredient, and, in addition, the quantity or proportion of certain specified substances.

The act in general requires the labeling of drugs and devices to bear warnings against probable misuse which may be dangerous to health. It also requires special precautionary labeling for drugs that are liable to deterioration. The act also prohibits interstate traffic in drugs which have been prepared or handled under insanitary conditions whereby they may have been contaminated with filth, or that may render the contents injurious to health. Those drugs which are official must be labeled as prescribed by the United States Pharmacopoeia and the National Formulary. If it purports to be, or is represented as, a drug the name of which is recognized in the United States Pharmacopoeia or the National Formulary, and its strength differs from, or its quality or purity falls below, the standard set forth in such compendium, the act requires its difference in strength, quality or purity from such standard to be stated plainly on its label.

Other Features

One of the most important features of the new act, from the standpoint of therapeutic claims, is that a drug or device is

misbranded if its labeling is false or misleading in any particular. When this act in general becomes effective, June 25, 1939, it will no longer be required by the government to prove that false claims of curative effect on the labels of drugs were made with willful intent to deceive, as required under the present Food and Drugs Act.

Criminal penalties under the new act have been increased as compared with the present act. It provides imprisonment for not more than one year, or a fine of not more than $1,000, or both such imprisonment and fine. Under a second conviction, the act provides for imprisonment of not more than three years, or a fine of not more than $10,000, or both such imprisonment and fine. However, if a violation involves intent to defraud or mislead, the penalty is imprisonment for not more than three years, or a fine of not more than $10,000, or both such imprisonment and fine.

RESTRAINT BY INJUNCTION

The new act limits seizure for misbranding to a single interstate shipment of the product, unless the misbranding has been the subject of a prior court decision in favor of the government, or unless the misbranded article is dangerous to health, or its labeling is fraudulent, or would be in a material respect misleading, to the injury or damage of the purchaser or consumer. However, the new act for the first time authorizes the federal courts to restrain violations by injunction.

No attempt has been made here to cover all of the requirements of the new act as they apply to veterinary remedies. More specific information may be obtained by consulting a copy of the act, which may be secured from the Food and Drug Administration, United States Department of Agriculture, Washington, D. C. Copies of a digest, which points out the differences between the new act and the present act of 1906, may also be obtained upon request.

By doing good with his money, a man, as it were, stamps the image of God upon it, and makes it pass current for the merchandise of heaven.—*Rutledge.*

Simple Shrinkage Test for Wool Fleeces Sought

From 30 to 70 per cent of the weight of wool fleeces as they are shorn from the sheep is foreign matter. The Bureau of Agricultural Economics points out that the wide variation in the quantity of clean wool a fleece will produce after it has been scoured makes it difficult to judge the market value of grease wool.

Under present methods of buying at country points, each buyer makes his own appraisal of shrinkage by the general appearance or "feel" of the wool. The Bureau says that such an appraisal is little more than a guess and places the producer at a disadvantage, since he is usually less competent than the buyer in estimating shrinkage correctly.

To make it easier for both the producer and the buyer to judge the market value of fleece wools better, government research workers, with the aid of a newly equipped wool-scouring laboratory at Washington, are attempting to develop a simplified method for determining "shrinkage." They are trying to develop a method for determining, from samples, the actual shrinkage that will take place in large lots of fleece wools.

In the past, sample tests have varied widely from the actual fiber outturn of larger lots. It is to perfect sample tests so that they will represent actual shrinkage, or bear a constant relationship to actual shrinkage, that these scouring tests have been undertaken.

Forest Tip from Dairyman

Owners of farm woodlands can take a tip from dairymen on ways to keep up production, says the United States Forest Service. The timber-owner needs to choose and keep his thrifty young trees just as carefully as the dairyman selects and keeps his best young heifers. When he culls out less thrifty timber, the better young trees grow faster. Cull trees can be sold as pulpwood, posts, or fuel wood. The thrifty trees left growing produce a more profitable crop later on.

Nonspecific Skin Diseases of the Dog*

By CARL F. SCHLOTTHAUER, *Rochester, Minn.*
Division of Experimental Medicine, The Mayo Foundation

Hardenbergh[1] has previously called attention to the fact that the skin of animals, with its protective covering of hair, is normally quite resistant to ordinary, injurious influences and that it also is endowed with peculiar powers of regeneration and repair. Slightly injured portions or areas heal without apparent inconvenience. This is necessary in order to enable the skin to carry out its chief functions: that of being a protective covering for the body and an organ of elimination.

CONTRIBUTING FACTORS

The skin is subjected to a variety of influences both from within and from without. Factors that influence the skin from within include improper diet, disturbances of the nervous and circulatory systems, digestive disorders, various infectious diseases, and certain drugs and chemicals. In the last named group should be included certain substances similar to hematoporphyrin which, if injected or ingested, make some light-skinned animals sensitive to light of certain wave lengths. Severe blistering may result if such sensitized animals are exposed to sunlight. Numerous external factors, such as trauma, extremes of heat and cold, chemical irritants, soaps, too frequent bathing, certain plant toxins and various animal and vegetable parasites, are capable of influencing the health of the skin. These agents may cause severe primary lesions in the skin and may progress to such an extent as to endanger the life of the animal.

CLASSIFICATION

Diseases of the skin are commonly grouped grossly according to their primary causes as parasitic and nonparasitic diseases. The latter group may be further divided into contagious and noncontagious conditions. The nonparasitic diseases of the skin are variously classified according to the lesions noted, symptoms manifested and their etiology. Unfortunately this last is too frequently unknown. Milks[2] stated that, during a ten-year period, 2,479 small animals affected with skin diseases were observed at the small-animal clinic at Cornell University; in about 42 per cent of these cases the cause of the disease was unknown. This agrees closely with my observations. This discussion will deal with some of these nonspecific skin diseases as they occur in dogs.

Nonparasitic skin diseases are commonly referred to as dermatitis or eczema. Haigler[3] recently suggested that veterinarians should use more specific and descriptive terms in describing lesions of the skin, and he suggested the more frequent use of the terms "erythematous," "exfoliative," "vesicular," "papular" and "pustular" in describing dermatitis. We all know that the term "alopecia" means "loss of hair," but alopecia areata and alopecia universalis are vastly different conditions. The general use of more descriptive and specific terms is indeed desirable.

ETIOLOGY

The etiology of diseases of the skin is so various that it is not always possible definitely to diagnose the primary disease in this organ. Not infrequently the lesion in the skin is secondary to disease in some remote part of the body, and it may be impossible to locate the primary lesion definitely. This undoubtedly is the reason why dogs occasionally are observed to irritate or mutilate grossly normal-appearing skin over certain areas of the body or limbs by biting or scratching. Khuen[4] called attention to the frequent association of acute, moist eczema areata in the sacral region and disease of the anal sinuses. In these cases treatment of the primary lesion in the anal sinuses usually is sufficient to promote healing of the skin condition. Hyperplasia of the glandular tissue surrounding the anus is frequent in old dogs. Undoubtedly this

*Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

too, can cause symptoms simulating inflammation of the anal sinuses. Myelitis can cause numbness or other irritating sensations in the limbs and body. A tendency to mutilate the hind legs, tail and perineum by biting was noted by me in three dogs affected with definite primary lesions in the central nervous system. In two of these cases, there was marked hydrocephalus with hydromyelia, and the third dog was affected with progressive ascending necrosis of the spinal cord.

NONSPECIFIC DERMATITIS

The skin diseases of unknown etiology, commonly referred to as nonspecific "eczema" or "dermatitis," without doubt cause veterinarians the most concern. There is one skin disease with which, I feel certain, many veterinarians are familiar. It is a dry, follicular dermatitis occurring chiefly on the backs and in the arm pits of coarse or wire-haired breeds of dogs during the summer months. This disease closely resembles follicular mange, but the presence of *Demodex folliculorum*, variety *canis*, cannot be demonstrated. The lesion commonly causes more discomfort than does follicular mange, dogs not infrequently scratching or biting the affected areas sufficient to cause bleeding and exudation. The disease commonly appears during the spring or early summer months and may persist until some time in the fall, when it will gradually disappear regardless of the kind of treatment applied. The lesions are very resistant to most remedies and usually do not respond satisfactorily to any treatment unless the animal is hospitalized; the lesions generally recur soon after the dog returns to his normal environment.

This disease may be due to various agents. Infected stripping combs and brushes, soaps, flea repellents, certain diets, allergy, photosensitization, certain plant toxins, algae, fungi and numerous other agents have been considered to be of etiologic significance. I have even heard mentioned the theory that the follicular irritation in some cases may be due to, or is associated with, the shedding of abnormal coarse or stiff hairs. Of these possible etiologic agents, improper diet and allergy have received the greatest attention in recent years. Burns,[5] Schnelle,[6] and Pomeroy[7] tested dogs for sensitivity to certain foodstuffs. I shall not report their results in detail, but they failed to show the major cause of these skin lesions. Schnelle was able to produce typical eczema in two dogs by feeding them substances to which they were sensitive. Leasure and Frick[8] recently reported the occurrence of allergic dermatitis in cattle. Coca,[9] in a recent report, stated that he did not think that the dermatitis of dogs was due to allergy. Some cases may be related to light sensitization, but it would be difficult indeed to associate the periodic or seasonal occurrence of this skin disease with food allergy. The dogs in our kennels receive various diets, but I have no record of the occurrence of a similar cyclic skin disease in these dogs.

SUMMER ECZEMA

The peculiar summer occurrence of nonspecific eczema or dermatitis suggests that the etiologic agent may be a parasite that exists in the outdoors or in certain plants, algae, or fungi. Cases observed by me would tend to suggest parasites that exist in the soil, or on the vegetation or certain algae. I have observed dogs whose skin would remain in normal condition as long as they were restrained from bathing in certain streams or lakes. In these cases I suspected the presence of some water-borne parasite or plant, although the etiologic agent may well have been on the shore. A well-known professional dog handler mentioned to me that she successfully treated a similar condition with sulfur baths. I have always been of the opinion that certain commercially-prepared remedies containing rotenone are beneficial. I have also used ointments containing sulfur and chaulmoogra, and they have likewise been beneficial in some cases. However, it has been observed that nearly all animals recover in the fall, regardless of the kind of treatment employed.

Dr. Theobald, of Cincinnati, observed similar cases and must be credited with making an important observation regarding the possible etiology of this skin disease. He noted that the condition occurred chiefly in chigger-infested (*Trombidium holoseri-*

ceum) areas and he was able to demonstrate the mite on some dogs. It would be unwise, however, to suggest that the chigger mite is the cause in all cases of similar nonspecific skin disease that occur during the summer months, although I am of the opinion that it is of importance. This would explain the seasonal occurrence of certain skin lesions that resemble follicular mange but in which *Demodex folliculorum* cannot be demonstrated.

OTHER CONDITIONS

There are, of course, many other kinds of nonspecific skin diseases. Furunculosis, alopecia, folliculitis and seborrhea in its various forms are not infrequent in dogs. The etiologic factors in these diseases are various and frequently are unknown, and I shall therefore not attempt to discuss them in detail. It has been my observation that these ailments occur more frequently in dogs that are maintained as house pets than in kennel-raised dogs. Undoubtedly diet, irregular exercise, too frequent bathing, certain soaps, flea repellents, and chemicals all play an important rôle. Various general diseases, and some internal parasites (chiefly hookworms), may cause dandruff, thinning of the hair and alopecia without causing gross changes in the skin. Any environment, diet, or disease that affects the general metabolism of an animal usually manifests itself in the skin and hair.

TREATMENT

Nutritional disturbances reduce the natural resistance of the skin and make it more susceptible to external harmful influences. Because of this it is frequently advisable to change the animal's diet and environment. Occasionally it is advisable also to bathe the animal to remove all evidence of previously applied remedies and débris and then observe him for two or three days to study the natural course of the disease. I commonly use a parasiticide in the bath. Quite often this treatment is all that is required. If further treatment is necessary, I usually use ointments or oily preparations if the lesion is dry and scaly, and drying lotions and antiseptic powders if the lesion is moist. I do not advise the use of one preparation for all conditions. One should always avoid too vigorous treatment and allow ample time for the skin to exercise its natural powers of repair between treatments.

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Federal Chemists Devise Quick Test for Food Cheat

A quick and cheap test is a first class aid to the Food and Drug Administration in protecting the public from food and drug cheaters. A simple test for mineral oil in butter—worked out by chemists of the Administration's Chicago laboratory—makes it easy to detect as little as 0.5 per cent of mineral oil, takes only about 20 minutes, and costs but a few cents for chemicals.

In the test the butter is melted, treated with a chemical, and then diluted with distilled water. A resulting milky or turbid solution indicates adulteration with mineral oil. Pure butter-fat is clear upon dilution.

On the rare occasions when butter is adulterated with mineral oil, the cheater stands to make a substantial profit from the substitution, at the expense of the consumer. Mineral oil sells for only a few cents a pound.

In the first big application of this new method, the Chicago office tested the whole output of one suspected plant—about $50,000 worth of butter in all. Some was of market quality, some lots were adulterated with mineral oil, and some short in butter-fat. The adulterated butter had to go to soap manufacturers. Butter low in fat had to be reworked to get rid of the extra water.

Vitamins in the Nutrition of Animals*

By WALTER C. RUSSELL, *New Brunswick, N. J.*
New Jersey Agricultural Experiment Station and Rutgers University

The word vitamin was coined in 1912, as a name for substances which an investigator by the name of Funk predicted would be found in foods. He visioned these substances as the preventives of certain age-old, human diseases such as beri-beri, scurvy, rickets and pellagra. It is likely that he did not have domestic species in mind during these early speculations.

A vast amount of research has been done in the vitamin field in the past quarter-century and we now know that the vitamins play a vital rôle in animal as well as human nutrition.

For many domestic animals, man now selects the food supply and such animals no longer have free choice of a presumably adequate diet. Therefore, a complete knowledge of the nutritive requirements of the domestic species is necessary for their health and for the efficient use of these species.

REGULATORS OF METABOLISM

From the chemical viewpoint all foods consist of molecules. Starch, fat and protein molecules have long been known but we now find it necessary to recognize a number of less well-known molecules in foods and among these are the vitamins. It may be trite to say this, but I should like to emphasize that everything that passes into the mouth of an animal is chemical in nature. Once in the digestive tract, a part passes into the blood-stream and the cell mass of the body, and the remainder becomes a part of the fecal material. The portion which reaches the cell mass may be used for tissue building or for the production of heat and energy, and certain of the molecules play the rôle of regulators of cell chemistry, that is, of metabolism. The vitamins, thus far studied, belong in the group of regulators of metabolism.

It is my plan to present to you, as concisely as possible, the present status of our knowledge of the nature of the vitamins, of their rôle in the animal organism and of their relation to certain disease conditions.

More than 25 substances have been reported as vitamins in the past 25 years. Thirteen are now definitely recognized, the formulas of seven have been determined and six have been synthesized in chemical laboratories. No chemical relationships are apparent among the formulas which have been worked out for the vitamins to date. Hence, we are not dealing with a class of substances of like chemical nature.

VALUES EXPRESSED IN UNITS

Only recently has it been possible actually to weigh vitamins and administer an exact quantity. In actual practice this can now be done with vitamins B_1 and C, riboflavin, nicotinic acid and carotene. Ultimately this will be possible with all of the vitamins. Heretofore, and even yet in the case of the factors which have not been isolated in pure form, it has been necessary to express vitamin values in terms of units. For the principal vitamins a League of Nations committee has defined a set of International Units and in the United States two legal units have been recognized by the United States Pharmacopeia Committee. These are for vitamins A and D in cod-liver oil and are called U.S.P. units. Theoretically they are equivalent to the International Units but in actual practice this relation does not hold, particularly with regard to vitamin A.

Our knowledge of the vitamin requirements of various species has grown rapidly and in table I an attempt has been made to show the vitamins necessary in the food of common species. To determine whether a species requires a given factor, it is

*Journal series paper of the New Jersey Agricultural Experiment Station, Department of Agricultural Biochemistry. Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

necessary first to devise a ration, complete in all respects except the factor under consideration, allow the deficiency to develop and then ascertain whether the feeding of the pure vitamin, a concentrate containing it or certain foods will bring about a cure. The most reliable results are obtained when tests can be conducted with pure vitamins. It should be emphasized that the zero (0) sign used in the table means that either no information is available or that the data are insufficient to permit a conclusion as to whether the vitamin is or is not required. For the turkey, duck, canary and parrot, our information is quite incomplete but it is likely that their requirements will be found to be essentially the same as those of the chicken.

able. When the white rat is on a fat-low ration, the vitamin-B_1 requirement is much higher than when fed a high-fat ration. Furthermore, the physical condition and activity of an animal may be important in arriving at an estimate of requirements. Thus, an active, high-strung type of dog may have a higher vitamin-B_1 requirement than a more lethargic type.

At this point the importance of an exact understanding of the meaning of the work requirement should be emphasized. For example, the fact that the cow requires vitamin A does not mean that a special vitamin-A preparation must be purchased. The usual constituents of a dairy ration supply an adequate amount of this factor. If a vitamin deficiency does appear, it is usually

TABLE I—*Vitamins required in the food of common species.*

SPECIES	VITAMIN							
	A	B_1	C	D	E	RIBOFLAVIN	NICOTINIC ACID	K
Chicken	+	+	—	+	+	+	—	+
Rat	+	+	—	+	+	+	0	0
Dog	+	+	—	+	0	+	+	0
Cattle	+	—	—	+	0	0	0	0
Pig	+	0	—	+	0	0	+	0
Turkey	+	0	0	+	0	+	0	0
Horse	+	0	0	+	0	0	0	0
Sheep	+	0	0	+	0	0	0	0
Duck	0	0	0	+	0	0	0	+
Goat	0	0	0	0	—	0	0	0
Cat	+	0	0	+	0	0	0	0
Canary	0	0	0	0	0	0	0	+
Parrot	0	0	0	0	0	0	0	0

+ = Required in the ration.
— = Not required in the ration but may be synthesized in the animal body.
0 = Either no information or insufficient data to warrant a conclusion.

QUALITATIVE INFORMATION

In the case of some species, particularly those of interest in agriculture, quantitative estimates have been made but much of our information is still qualitative in nature. As we learn more about the interdependence of nutritive factors, it may develop that a quantitative requirement cannot be given except as related to the other constituents of the ration. Thus in the growing chicken, leg-weakness can be prevented by feeding 20 U.S.P. units of vitamin D per 100 grams of feed; but only if adequate calcium and phosphorus are available. When the white rat is on a fat-low ration, the vitamin-B_1 requirement is much higher than when fed a high-fat ration. Furthermore, the physical condition and activity of an animal may be important in arriving at an estimate of requirements. Thus, an active, high-strung type of dog may have a higher vitamin-B_1 requirement than a more lethargic type.

At this point the importance of an exact understanding of the meaning of the work requirement should be emphasized. For example, the fact that the cow requires vitamin A does not mean that a special vitamin-A preparation must be purchased. The usual constituents of a dairy ration supply an adequate amount of this factor. If a vitamin deficiency does appear, it is usually better and more economical to correct it with common feeding materials than to purchase a special product, which is frequently labeled with a fancy trade name.

Ordinarily the common feeding stuffs contain all of the known vitamins with the exception of vitamin D. However, if the products used in the compounding of rations have been subjected to processing or if the finished product is processed, as in the case of dog foods, there is the possibility of the destruction of vitamins unless the processing conditions are known and controlled. It is more important, therefore,

to know the vitamin content of a product as it enters an animal's mouth than in its original state.

The present status of our knowledge of the better known vitamins and of their rôle in animal nutrition can best be presented by a consideration of each of the vitamins separately. For convenience let us consider them in alphabetical order.

VITAMIN A

Vitamin A occurs in two forms, a provitamin form and the vitamin itself. The provitamin form is the yellow plant pigment, carotene. This substance is converted to vitamin A by the animal organism and apparently all of the common species, with the possible exception of the cat, are able to effect this conversion. It is of interest in this connection that the dairy breeds which produce the colorless or light-colored milks are better converters of carotene to vitamin A than are those which produce a pigmented milk. Thus the Holstein cow is a better converter of carotene to vitamin A than the Jersey. On the same ration, however, the vitamin-A potencies of the two types of milk are essentially the same.

The exact chemical function of vitamin A in the organism is as yet unknown. A lack of the vitamin in the white rat results in the replacement of the normal epithelium of the respiratory, digestive and genito-urinary tracts with a cornified or keratinized squamous epithelium. This abnormal epithelium is readily invaded by disease organisms and, on account of this observation, vitamin A became known as the anti-infective vitamin. It is an anti-infective vitamin to the extent that when enough is present to maintain normal epithelial linings the chances of invasion by disease organisms are lessened, but no satisfactory evidence has ever been presented that the feeding of an excess of this factor develops a still greater resistance to disease invasion. Vitamin A is not unique as an anti-infective agent, because a lack of other nutritive factors, even when plenty of vitamin A is present, will produce a deficiency condition which permits an easier invasion by disease organisms than in the normal animal.

The visual purple of the retina is formed from vitamin A. Night blindness in the human, that is, the inability to see well at night, is said to be due to an inadequate amount of vitamin A for the formation of visual purple. Night blindness in horses, for example, may thus be due to an inadequate amount of vitamin A in the ration, and this lack is quite likely to occur when the horse is fed largely on field-cured hay and oats, both of which are notoriously low in this factor. Night prowling animals such as the dog and cat probably have rather high vitamin-A requirements because of the need for visual purple.

A frequent manifestation of a lack of vitamin A, especially in a severe deficiency, is metaplasia of the epithelium of the conjunctiva and cornea. Infection sets in and in some cases the cornea becomes ulcerated.

The best source of vitamin A for farm animals is the carotene of green plants, yellow roots and yellow corn. In the case of yellow corn, however, the provitamin is in part cryptoxanthin, a pigment similar to carotene. Summer pasturage supplies plenty of carotene but this pigment is largely destroyed by the field-drying conditions which prevail during the haying season in many parts of the country. Artificial drying of forage crops and special methods of ensiling grasses and legumes preserve the vitamin A in crops stored for winter use.

For carnivorous animals liver and other glandular organs are an excellent source of the vitamin, but muscle meat is practically devoid of the factor. Fish-liver oils and fish-liver-oil concentrates also are rich sources and these along with carotene are used extensively in prepared foods, such as dog and cat foods. For the farmer it is cheaper to produce or purchase his own supply of vitamin A in the form of carotene-rich crops, and preserve them well for winter use, than to buy special vitamin-A or carotene preparations.

Both carotene and vitamin A have been isolated, their molecular structures determined and the synthesis of vitamin A has been reported. Now that the isolated vitamin is available, the most important advance that can be made in our knowledge of this factor will be the determination of

its specific rôle in the chemical actions in the animal organism.

VITAMIN B

Early in the history of vitamin research, it became evident that what was at first called vitamin B was a complex of several factors. At one time, no less than 17 vitamins B were listed. An aggressive attack by a number of research workers has resulted in the isolation, mapping of chemical structure and synthesis of vitamin B, known also as B_1, and now called officially thiamin chloride. Another fraction, B_2 or G, has been isolated and found to be riboflavin. It also has been synthesized. For a while, vitamin G was called the anti-pellagra factor and was thought to prevent blacktongue in dogs and pellagra in the human. Recently the anti-pellagra and blacktongue-prevention factor was found to be nicotinic acid. Therefore, three members of the vitamin-B complex are now definitely known compounds.

An adequate supply of the vitamin-B group of factors for the common species, having free access to the ordinary feeds, has never been considered a serious problem. What we have called vitamins B and G have quite a widespread occurrence in cereals, forage crops, silages and skimmed milk; and in adequate potencies unless processing of these products has been too severe and the vitamins have been removed or destroyed. Vitamin-B_1 deficiency has not been a prominent nutritional disorder except in animals, such as the dog, subsisting on prepared foods of poor quality or which have been too highly processed.

A deficiency of this vitamin can readily be produced in the dog and the chicken. Failure to grow, weakness, and polyneuritis in the extreme deficiency are the outward manifestations. The specific rôle of this factor in metabolism is that it forms a part of the molecule of cocarboxylase which is necessary for the removal of pyruvic acid, a product formed during the utilization of glucose in the animal organism. The primary effect of a lack of vitamin B_1 is, then, the accumulation of pyruvic acid, which is an interference with normal utilization of glucose.

Growth failure, weakness and polyneuritis are probably secondary effects and general manifestations of this particular type of disturbance of carbohydrate metabolism. The discovery of the rôle of vitamin B_1 is the first instance of a clean-cut picture of the fundamental rôle of a vitamin in the animal organism. The sooner basic information of this type is obtained for the other vitamins, the more rapid will be our advances in their use in human and animal nutrition.

Certain species, the ruminants, may not require a supply of vitamin B_1 in the ration. Evidence points to the formation of this factor by bacteria in the rumen of the cow, for example.

RIBOFLAVIN

Riboflavin, formerly called vitamin G, is widely distributed in plant and animal tissue. Combined with phosphoric acid and a protein it forms a yellow oxidation pigment or enzyme which may be present in all living cells, at least those of the higher forms of life, and has to do with cell respiration. Thus, riboflavin, like vitamin B_1, is necessary in the enzyme systems which catalyze the chemical reactions of the body.

On a ration deficient in riboflavin, chicks fail to grow normally and develop a characteristic leg disorder. Also, when the level of this substance is low in the ration of the hen, there is interference with hatchability. In the dog a lack of riboflavin results in a symptom complex which includes a yellow mottling of the liver and degenerative changes in the central nervous system. Some investigators have reported cataract formation in the eyes of rats and chickens on experimental rations deficient in riboflavin. Whether this condition can be produced in other common species remains yet to be demonstrated.

NICOTINIC ACID

It should be emphasized again that riboflavin (vitamin G) will not cure pellagra in the human or blacktongue in the dog. Within the past year, Elvehjem, at the University of Wisconsin, has made the outstanding discovery that a rather common organic compound, nicotinic acid, will cure

or prevent blacktongue in the dog and pellagra in the human. Although nicotinic acid can be prepared from nicotine in the laboratory, nicotine itself has no effect on pellagra or blacktongue. Certain foods, such as milk, fresh vegetables, eggs and glandular organs, such as liver, will cure or prevent these deficiencies, as will yeast, and it is interesting to speculate concerning the origin of nicotinic acid in these substances.

The anti-pellagra or anti-blacktongue factor was never given an alphabetical designation, as has been the case with the other vitamins. In fact the actual substance was discovered before a letter was assigned—so are we going to call nicotinic acid a vitamin? The situation raises the interesting question as to just what substances should be called vitamins—and what does the term really mean? The domestic pig also requires this substance, but whether it is necessary for species other than man, the dog and the pig has yet to be ascertained.

Only mere mention can be made of other factors of the B complex. Vitamin B_3 is necessary for pigeons, B_4 for the prevention of a specific paralysis in rats and chicks, B_5 for weight maintenance in pigeons, B_6, or H, for the prevention of a nutritional dermatosis in rats and W for growth in rats. None of these, with the exception of B_4 for chicks and the probability that B_6 is necessary for dogs, has been demonstrated as being required by the common domestic species.

VITAMIN C

Only man, monkey and the guinea pig require vitamin C in their food. The common domestic species apparently form this factor in their bodies, and consequently it is of little moment in this discussion. The chemical structure of this vitamin is known, it has been synthesized, and the synthetic form may be purchased as ascorbic or cevitamic acid. Chemically it resembles the simple sugars. The common sources are fruits and vegetables, particularly the citrus fruits.

Except for vitamin A, which has been widely advertised, but erroneously, as the anti-infective vitamin, vitamin D, the anti-rickets vitamin, has probably been the most widely publicized factor. The fact that this vitamin can be developed in foods by exposure to the ultra-violet rays of an artificial source or of sunlight has led to the designation, "the sunshine vitamin." This has caught the public imagination and products made antirachitic by irradiation with ultra-violet light are now well known. The substances activated are the sterols of foods, and of the skin when the animal body is irradiated, such as certain forms of cholesterol and ergosterol. Research has been very active in this field, partly because this is probably the only vitamin that one really needs to purchase in the feed store, grocery, or drug store. Obviously large financial returns are to be expected from the production and sale of this vitamin, and rightly so.

VITAMIN D

The very active research in this field has brought to light six and possibly eight different vitamins D. Fundamentally they have the same chemical architecture and differ only in certain portions of the molecule.

This vitamin seems to be required by all of the common species, but according to our present information needs to be added to the ration only in the case of those species which do not ordinarily have free access to sunlight; especially during the period of pregnancy and rapid growth when bone formation is going on actively. This is the only vitamin which is known to require the presence of other substances in the dietary in order to be effective. Adequate supplies of calcium and phosphorus are necessary along with vitamin D for normal bone and tooth formation.

The best natural source is summer sunlight, although winter sunlight has considerable potency unless there is absorption of the ultra-violet rays by smoke and fog. None of the common feeds contains the factor in an appreciable amount, but the fish-liver oils are a rich source. You are undoubtedly aware of the fact that the baby-chick industry, which can now supply chicks at any time of the year, is depend-

ent largely upon a scheme of management which permits the growing of the chicks to the broiler or laying stage, and even for use as laying birds, indoors. This management scheme has been made possible by the use of fish-liver oils as a source of vitamin D.

Some of the vitamins D show specificity toward certain species. The outstanding case is the different responses of the rat and the chicken to irradiated ergosterol, sold as the pharmaceutical preparation viosterol. Vitamin D in the form of cod-liver oil is equally effective in the rat and chick. When vitamin D is fed as irradiated ergosterol, however, 150 to 200 times as many units of the vitamin are required by the chick as are necessary in the form of cod-liver oil. This is a marked difference in species response to two forms of vitamin D. As work progresses it is possible that other species differences will be found.

VITAMIN E

In 1922, the discovery of a factor necessary for reproduction in rats was announced. White rats on an otherwise complete ration failed to reproduce unless this factor, called vitamin E, was supplied. A lack of almost any essential nutritive factor will interfere with normal reproduction, as will certain disturbances of the normal physiology of an animal, such as those of the endocrine system. Hence, reproductive failure due to a deficiency of vitamin E is not unique but the manner in which reproduction is affected is unique.

The rôle of vitamin E has been studied extensively in the white rat and the sequence of events is well known. Fertilization of the egg and implantation of the fertilized egg take place and the fetus grows for about one-quarter of the gestation period. Then growth becomes slower, the fetus degenerates and absorption follows. When an adequate quantity of the vitamin is administered to the female and she is again mated, a normal pregnancy ensues.

A lack of this factor apparently interferes with the normal, rapid proliferation and differentiation of cells in the fetus. The vitamin-E-deficient female rat was certainly not sterile in the usual sense or fertilization of the ova would not have occurred. Hence, to call vitamin E the antisterility vitamin is not an accurate description of its function. Permanent interference with reproduction does not result in the female white rat, but complete suspension of spermatogenesis occurs in the male when deprivation is too prolonged.

In the domestic species, extensive, controlled experiments have been conducted only in the case of goats and chickens. Thomas, at the Iowa Experiment Station, has shown conclusively that reproduction in goats is not disturbed when they are fed a ration which is sufficiently lacking in vitamin E to prevent reproduction in the rat, and several investigators have shown that the chicken requires this vitamin.

Adequately controlled experiments have not been conducted with cows, horses, sheep and pigs. In Denmark a large percentage of successes has been reported as resulting from the injection of a vitamin-E concentrate into dairy cows which have come into heat regularly but in which the fetus has failed to develop. These results have not been confirmed by other workers. At the present time, there is no adequate experimental evidence that vitamin-E preparations should be purchased by the dairy farmer to be added to the ordinary ration of the dairy cow or administered by injection. An experiment to determine whether vitamin E is necessary for reproduction in the cow would be a very expensive undertaking because of the difficulty involved in obtaining a ration deficient in vitamin E, yet adequate in other respects.

The occurrence of this vitamin is widespread in the usual feeding materials. In the cereal grains and other seeds it is concentrated in the germ. Wheat-germ oil, for example, is an especially rich source. Extensive degermination of cereals reduces the vitamin-E content, but well-preserved forage crops used in the ration probably supply an adequate amount. Emphasis should be placed on the fact that although wheat-germ oil is rich in vitamin E, it also contains other substances and that the results which have been reported with domestic species are due to wheat-germ oil and not isolated vitamin E.

In the case of the other large domestic mammals, the horse, pig and sheep, and also the dog and cat, no evidence of a satisfactory nature has been obtained that vitamin E is necessary for these species or should be added to the commonly used feeding materials. A lack of the vitamin in laying rations for hens inhibits hatchability, but the usual rations apparently contain adequate amounts of the factor.

The vitamin has been isolated and recently it was synthesized. A satisfactory method of assay has not been developed and a generally accepted unit has not been established.

No Vitamin F

Despite claims to the contrary, a vitamin designated by the letter F is not recognized by research workers in this field. At one time this letter was proposed for what is now B_1 but its use was never widespread. In 1934, the term vitamin F was suggested for the fatty acids, linoleic and linolenic, essential in the nutrition of the rat. Although the necessity for these substances has never been demonstrated in any other species, attempts have been made to promote their use. In recent months, the American Institute of Nutrition approved a recommendation that the term "vitamin F" not be used in referring to any fatty acids or mixtures of fatty acids.

Within recent years, a factor designated vitamin K has been discovered which is necessary for normal coagulation of blood in the chicken. Absence of this factor results in a development of hemorrhages. As yet studies have been made only with the chicken. The vitamin occurs in certain animal fats, leafy vegetables, fish meal, and cereals to some extent. So far this deficiency has not been observed on the usual poultry rations.

Up to this point, I have endeavored to give you basic, fundamental information concerning the vitamins, and it now seems in order to raise the question as to what you, as practicing veterinarians, would like to know about the use of these substances.

Vitamins Highly Specific

The simplest and most fundamental fact that can be stated with regard to the relation of vitamins to disease is that a given vitamin will cure only the disease condition caused by a lack of that vitamin. In their action the vitamins are highly specific. One vitamin will not cure a deficiency caused by the lack of another. Furthermore they are not cure-alls, as some would lead us to believe.

The external changes which take place in an animal in a vitamin-deficient condition are the result of a disturbance of the normal chemistry of the cells. As this disturbance becomes progressively more severe, the external symptoms develop. These external symptoms might conceivably lead to a diagnosis as one disease at one stage of the development and as another at a later stage. Thus the failure to eat and lassitude of a dog, at one stage in the devolpment of vitamin-B_1 deficiency, might be taken for distemper and at a later stage the posterior paralysis might be diagnosed as a toxemia. Names have usually been assigned to disease syndromes even before the disease has been clearly described or its etiology known. Hence certain diseases, already named, but of obscure etiology, may be due to a vitamin deficiency.

The only way in which it can be demonstrated with certainty that a vitamin will cure a disease condition is first to produce the disease on a dietary which lacks only the vitamin under consideration and then bring the experimental subject back to normal by the administration of the pure vitamin, not a concentrate containing it. Unless a vitamin has been isolated in pure form, it is obvious that it cannot be used for such a test. Furthermore, results obtained with one species may not be applicable to others. Many studies have been conducted with that indispensable laboratory animal, the white rat, but it does not follow that the results will apply directly in other species. The only reliable information for use with a given species is that obtained by experiments with the species itself.

EARLY DIAGNOSIS OF AVITAMINOSIS

One of the pressing problems in the nutrition field is the early diagnosis of vitamin and other nutritional deficiences. Before many years, rapid laboratory methods for this purpose may be available. At present, you see only the external indications, ordinarily after the deficiency has developed to a severe stage, and these may be secondary in character. Thus, the xerophthalmia seen in the cow, dog or chicken in a vitamin-A-deficient condition is secondary in character and is due to an invasion by microörganisms of the epithelial tissues which have been altered in structure by the lack of this vitamin.

Vitamin-A deficiency in the dog may be confused with distemper. In fact, vitamin-A deficiency could be confused with vitamin-B_1 deficiency in the growing puppy in the early stages, both deficiencies being marked by failure to eat and to grow at the normal rate. Chickens may "go down," as we say, because of weak legs. Is this due to a calcium or phosphorus deficiency, a lack of vitamin D or of some member of the vitamin-B complex? External appearance may not reveal the true cause and, with an inexpensive species like the chicken, it is possible to make certain laboratory tests on birds taken from the flock and killed.

Until satisfactory clinical methods are available, it will be necessary for the veterinarian to record the nutritional history of the subject under examination, determine whether one or more of the vitamins, or other dietary essentials, is lacking and prescribe accordingly. This procedure requires that the veterinarian become familiar with the nutritive properties of the various feeding materials. If there appears to be a lack of any of the vitamins, then the addition of foods rich in the missing factor is indicated and in a severe deficiency, especially in the case of dogs and cats or valuable, pedigreed farm stock, it may be advisable to administer the pure vitamin until the animal is able to eat normally.

SYMPTOMATOLOGY

Clean-cut, detailed descriptions of the progressive stages of vitamin deficiencies in the common species are as yet available in only a few cases. Some are available but time does not permit discussion of them. They will, of course, appear from time to time in your professional journals. Hart and Guilbert,[1, 2] of California, have already reported excellent descriptions of vitamin-A deficiency in certain domestic species. For the benefit of the practicing veterinarian such descriptions should be prepared as soon as possible.

There is little likelihood of any vitamin deficiencies in dairy animals, horses, sheep and goats on pasture, except during drouth. Likewise pigs, chickens, turkeys and ducks, with access to natural food supplies, and fed some grain, rarely show diseases traceable to the lack of vitamins. When man takes his domestic animals and pets away from their natural food supplies and environment, the vitamin and other dietary deficiencies begin to appear.

The cow, when barn fed, is likely to develop a vitamin-A deficiency if poor quality roughage, or an inadequate amount, is fed. Similarly the horse, sheep and goat may be affected. Unless the cereal grains are too highly processed, the members of the vitamin-B group required by these species are not likely to be lacking. The vitamin-D requirements of these species are not well known, but apparently are not high. However, very close confinement may lead to a deficiency of this factor. Swine reared indoors and pregnant and nursing sows may suffer from a vitamin-D deficiency, and lack of A is a possibility.

If the chicken is housed, it will soon develop a vitamin-D deficiency unless one of the fish-liver oils or fish-liver-oil concentrates is supplied. A vitamin-A deficiency may occur but this is not likely on a ration which contains yellow corn and dry or fresh green food. Deficiencies of other vitamins are rarely observed. What has been said with regard to the chicken probably applies equally well to the turkey, duck, canary bird and parrot.

The dog and cat being closer to man, and more dependent upon his individual ideas of a dietary than the other species, are more subject to dietary deficiencies. Little is known about cat nutrition, but in the

case of dogs attention should be given to the occurrence of deficiencies of vitamins A, B_1 and D, riboflavin and nicotinic acid.

As you well know, other dietary deficiencies may occur, such as mineral and protein deficiencies, so that the vitamins are not the only factors concerned in deficiency diseases.

The term vitamin was coined really to cloak our ignorance of the nature of these substances. As our knowledge of their chemical nature advances, we can discard the word vitamin. Thus research workers are already beginning to use the term thiamin chloride in place of vitamin B_1. There is nothing magic about the vitamins. They are simply chemical substances required for the normal chemical reactions of the animal organism.

This has been an attempt to summarize for you the present status of our knowledge of vitamins in the nutrition of animals. I know it has been inadequate, because our exact knowledge of the whole field is as yet inadequate. For the present the safest rule to follow is to feed yourselves and the animals under your care with as wide a variety of foods of good quality as the species in question will consume.

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Poor Trapping Makes Coyote a Real Pest to Farm Animals

On the western ranges, a four-legged coyote is a pest to stockmen, but a three-leged coyote is worse. The latter, called "peglegs" in that country, are those which have escaped from a trap with the loss of one foot—usually a fore foot.

Because they are handicapped in catching the swifter wild animals, such as jack rabbits and rodents, the coyotes prey on small domestic animals, such as sheep, goats and, occasionally, calves. Their handicap increases their boldness. The average coyote is a help to the farmer to the extent in which he feeds on rodents and rabbits. These make up about one-third the food of the average coyote, but only about one-fifth that of the "peglegs."

From these observations, the United States Biological Survey points out that the crippled coyote which escapes because of inexpert trapping and poor traps is more of a tax upon live stock than the coyote which has not been trapped.

The Biological Survey made a study of the stomachs of 161 "pegleg" coyotes. The results confirmed the prejudice against crippled coyotes long held by observant farmers, and showed the value of careful trapping. They found that animals such as sheep and goats—rarely calves, colts or hogs—accounted for 13½ per cent of the food of normal coyotes, but made up about 21 per cent of the diet of cripples.

Cosmetic Industry Is Heavy User of Beeswax

Lipstick, rouge, mascara, eye-shadow, face cream, and a goodly list of other beauty aids depend each year upon the busy bee for a million pounds or more of the products entering into their manufacture.

According to H. J. Clay, of the Bureau of Agricultural Economics, most present-day cosmetics contain beeswax as a part of their preparation. Fully 50 products used on the face and hands include some beeswax, but the chief use of this item is in cream and make-up, such as lipstick and rouge.

Typical cold cream contains from 8 to 12 per cent of beeswax. Deodorant sticks may contain as much as 35 per cent, camphor ice 25 per cent, and epilating wax 20 per cent. Cold cream, from a formula that has come down from the Roman era, includes beeswax.

According to Clay, no synthetic substitute has yet appeared which will adequately take the place of beeswax. The cosmetic industry now ranks second only to the production of special types of candles in the quantity of beeswax consumed.

Paratyphoid Infection of Turkeys*

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INTRODUCTION

During the past several years, organisms belonging to the paratyphoid group have been recovered not infrequently from the internal organs of young turkey poults. The records of the Diagnosis Laboratory at University Farm show that *Salmonella aertrycke* was first isolated from poults in May and June, 1932. The organism was recovered from four young turkeys that originated from two different ranches. During the period 1932-37, *S. aertrycke* type was recovered from poults which originated from 31 widely separated flocks. The losses from this infection were encountered during the months of April, May, June and July. The disease was acute, the losses varying from 10 to 25 per cent, and the infection appeared in poults varying from a few days old to five weeks. The most critical period was from ten days to two weeks of age.

The disease appeared on one farm in 1935 and recurred the next season. The breeding flock, consisting of 155 birds, was tested with whole-blood pullorum and aertrycke plate antigens in March, 1937; 13 (8.38 per cent) reacted and were removed from the flock. The disease did not appear in the poults during the 1937 season. The breeding flocks on three other farms where the disease had occurred during the preceding year were tested in March, 1937. The three flocks contained 686 birds. Fifty-five birds (8.01 per cent) reacted and were removed from the flock. The disease was not encountered in young poults reared on these farms during the hatching season.

The organisms isolated from the poults during 1935, 1936 and 1937 were kept for more critical study and examination. The morphological and cultural characteristics, as well as the carbohydrate reactions, of these organisms met the requirements of *S. aertrycke* as described by Bergey.[1] Edwards[2] found these strains to differ serologically.

LITERATURE

An extended review of the literature will not be included. Paratyphoid infections of young turkey poults and young chicks have been investigated by Rettger, Plastridge and Cameron,[3] Lee, Holm and Murray,[4] Cherrington, Gildow and Moore,[5] Schalm,[6] Jungherr and Clancy[7] and Edwards.[8,9] In these studies the authors have identified the causative organism as *S. aertrycke* or an organism of that type.

According to Schalm, the fumigation of the incubator by the cheesecloth method after one-tenth of the chicks had hatched and again from 12 to 15 hours later, appeared to control the spread of *S. aertrycke* infection.

DATA AND PROCEDURES

In July, 1935, the Diagnosis Laboratory at Minnesota began a study of paratyphoid infection of poults. The organisms isolated in 1935 were found to be pathogenic for guinea pigs, rabbits and young poults. Adult chickens were able to withstand intravenous injections. A questionnaire was sent to the flock-owners in an endeavor to obtain information regarding the course of the disease in the infected flocks. As a result of the survey, 584 adult birds were tested during January, 1936, with whole-blood pullorum and aertrycke plate antigens. Seven birds showed slight reactions to the plate tests. The blood-serums of this group of birds were tested by the test-tube method with eight Salmonella antigens, viz., *S. aertrycke, S. pullorum, S. gallinarum, S. suipestifer, S. abortus-equi, S. enteritidis, S. paratyphi A* and *S. paratyphi B.* As a result of the above tests, three turkey hens (512, 793 and 4728) were obtained for additional study. A turkey tom (485),

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whose blood-serum did not agglutinate any of the above antigens, was added to the group. In this study, unless otherwise stated, the following dilutions were used: 1:25, 1:50, 1:100, 1:250, 1:500 and 1:1000. The maximum titre of the blood-serum of each of the three hens, when tested with *S. aertrycke* antigen, was 1:100. With *S. pullorum* antigen the blood-serum of hen 512 was incomplete at 1:100; hen 793 had a titre of 1:500, and hen 4728 had a titre of 1:50. Agglutinins for *S. paratyphi A* antigen were found in the blood-serum of one bird (793). The titre was incomplete at 1:500. The blood-serum of the latter bird also agglutinated *S. gallinarum* antigen; the titre was incomplete at 1:250.

The Salmonella antigens were prepared as follows: The organisms were grown on beef infusion agar for 72 hours, and the growth was washed off with 0.5 per cent phenolized saline. The bacterial suspension was standardized to 0.04 per cent by the centrifuge method. The suspension was diluted with 0.3 per cent phenolized saline to make the desired concentration. Cultures of paratyphoid A and B were obtained from the Minnesota State Board of Health. The *S. enteritidis* culture was isolated from a rat. *S. suipestifer* was of swine origin, and *S. abortus-equi* was isolated from a mare. *S. gallinarum* and *S. pullorum* were isolated from chickens and *S. aertrycke* was obtained from turkeys.

The turkey hens (793, 512 and 4728) were trap-nested from March 26, 1936, to July 20, 1936. The eggs were collected three times daily and placed in the incubator at weekly intervals. The eggs were candled each week and all infertile eggs and dead embryo eggs were removed.

BACTERIOLOGICAL EXAMINATION OF EGGS

Mallman and Moore[10] reported the results of the comparative study of three methods of bacteriological examination of eggs for *S. pullorum*. In our study the following technic was used. The eggs were prepared for culturing by placing them in a 1:500 aqueous solution of bichloride of mercury for 30 minutes. The large end of the egg was heated with a flame, then the egg was opened with sterile forceps and the contents transferred to flasks containing about 100 cc of sterile beef infusion broth and incubated for 72 hours at 37.5° C. The broth was then streaked on plates containing agar to which had been added 10 per cent normal horse serum. Typical colonies were transferred to agar slants for further study and examination.

In the case of dead-in-the-shell poults, the embryos were opened aseptically and a portion of the liver was placed in beef infusion broth and incubated for 48 hours. The broth was then streaked on serum-agar plates. Typical colonies were transferred to serum-agar slants. The yolk sac was also cultured and handled in a like manner.

A total of 103 eggs were incubated. From this number, 14 living poults were hatched. Of the remaining number, 16 were infertile, 11 were broken in the incubator, 23 contained partially developed dead embryos and 39 had dead full-term embryos. The 78 eggs that failed to hatch were examined bacteriologically with the following results: *B. subtilis* was recovered from seven eggs, *E. coli* from four eggs, *Escherichia* sp. from three eggs, an unidentified staphylococcus from one egg, and 63 eggs were found sterile. The broken eggs were not examined.

BACTERIOLOGICAL EXAMINATION OF POULTS

The first death occurred on the third day, and two more poults died during the third week. Bacteriological examination of the liver, heart and lungs gave negative results.

Agglutination tests of the blood-serums of the eleven poults raised to maturity were made at monthly intervals. The serums were tested with the eight antigens mentioned previously. Negative results were obtained.

The tom in the original pen remained negative to the agglutination test for the entire five months of observation. The hens were destroyed at the end of the hatching period, and a bacteriological examination was made of the liver, heart, spleen, kidney, ovary, bone-marrow and intestine. *S. aertrycke* and *S. pullorum* were not recovered from the internal organs.

The studies were continued in 1937. Inquiries were sent to flock-owners who had encountered aertrycke infection in their young poults. Four flocks were available for study. These flocks were composed of birds that had been exposed to infection during the preceding year. The four flocks contained 841 birds. These birds were tested with the rapid whole-blood method, using *S. pullorum* and *S. aertrycke* as antigens. Agglutinins were found in the blood of 68 birds.

Blood was collected from the above birds, and the samples were numbered for convenience from 1 to 68, inclusive. The blood-serums were tested on March 15, 1937, with all antigens mentioned before. In this instance only three serum-antigen dilutions were used, viz., 1:25, 1:50 and 1:100. Of this group of serums, only six agglutinated aertrycke antigen in the highest dilution (1:100), while the maximum titre of 13 other serums was 1:50. All of the blood-serums contained agglutinins for pullorum antigen.*

Nine turkey hens were purchased from this group of birds (10, 22, 36, 41, 50, 57, 58, 60 and 66). With one exception (hen 22) the blood-serums of the above birds contained agglutinins for *S. aertrycke*. The blood-serums of hens 58 and 60 also agglutinated *S. gallinarum, S. paratyphi A* and *S. paratyphi B*. A tom whose blood-serum contained no agglutinins to these antigens was added. These birds were trap-nested from March 26 to July 22, inclusive. The eggs were collected three times daily and stored at a temperature of approximately 45°F. They were placed in an electric, forced-draft incubator each week, and the temperature was regulated between 99° and 101°F. The eggs were candled each week and infertile and dead-embryo eggs were removed. In all, 325 eggs were incubated. Of this number, 90 were found infertile at the end of the first week of incubation, 24 dead embryos were found at the end of the second week of incubation, 32 additional dead embryos were removed at the end of the third week of incubation and, at the end of the fourth week, 57 more dead embryos were found. Of the remaining 122 eggs, 115 living poults were hatched and the remaining seven eggs had cracked shells but the poults failed to emerge.

*The complete data relating to the agglutination test of these samples are available.

Bacteriological Examination of Eggs

Infertile eggs and dead embryos were examined bacteriologically as described before.

S. aertrycke was isolated from three infertile eggs, from one egg that contained a dead embryo at the end of 14 days of incubation, from two eggs that contained dead embryos at the end of 21 days of incubation, and from one egg that contained a dead poult. *E. coli* was recovered from 36 eggs, staphylococci from 27 eggs, Pseudomonas from three eggs, Gram-positive rods from 15, and 115 eggs were found sterile.

Examination of Poults

When poults were 24 to 36 hours old, they were placed in a room where the temperature was maintained between 95° and 100°F. Each hatch was confined to a separate wire cage. The birds were raised on wire netting until eight weeks of age. They were then tested with whole-blood pullorum and aertrycke plate antigens. No reactors were found. The poults then were transferred to a pen containing straw litter and allowed to mingle with other birds. From then on, all the birds were reared as one unit.

Eighty-eight of the 115 poults died or were destroyed because they were crippled. The internal organs (liver, heart, yolk sac, lungs and intestines) were examined bacteriologically. *S. newington* was isolated from three, and *S. montevideo* was isolated from one. Salmonella organisms were not recovered from the intestinal tract. *E. coli* and an organism belonging to the Pseudomonas group were frequently recovered.

Examination of the Adult Birds

During the summer of 1937, the parent birds were destroyed and blood was collected for agglutination testing purposes. Bacteriological examination was made of the liver, heart, spleen, kidney, ovary, ovi-

duct, intestine and bone-marrow. *S. pullorum* was not recovered in any instance. *S. aertrycke* type organism was recovered from the ovary only in birds 36 and 58. From bird 22 (when selected this bird was negative to the agglutination test), the organism was recovered from the ovary and the oviduct.

Maximum agglutination titres of the parent stock were ascertained by the test-tube method. The antigens previously mentioned were used. Only those antigens that were agglutinated will be mentioned and the maximum titre designated. Hen 10 was destroyed during September. The titre was 1:100 with aertrycke antigen, incomplete at 1:500 with pullorum antigen and 1:25 with paratyphi A antigen. Hen 22 had a negative titre to aertrycke antigen when originally selected, but developed an incomplete titre at 1:250 to that antigen when slaughtered during July. She also had an incomplete titre at 1:100 with pullorum antigen. Hen 36, destroyed in August, had an incomplete titre at 1:100 with aertrycke antigen. Hen 41, destroyed during August, had an incomplete titre at 1:50 with aertrycke antigen. Hen 50, destroyed in August, had an incomplete titre at 1:500 with aertrycke antigen, a titre at 1:250 with pullorum antigen, a titre at 1:50 with paratyphi A, incomplete at 1:50 with paratyphi B and a similar titre with gallinarum antigen. Hen 57, destroyed in September, had a titre at 1:50 with aertrycke, 1:1,000 with pullorum, 1:250 with paratyphi A, 1:1,000 with paratyphi B, 1:1,000 with enteritidis, and 1:1,000 with gallinarum antigen. Hen 58, destroyed during June, had an incomplete titre at 1:500 with aertrycke antigen. Hen 60, destroyed during July, had a complete titre at 1:100 with aertrycke and pullorum antigens and incomplete at 1:100 with gallinarum antigen. Hen 66, destroyed during May, had a 1:50 titre with aertrycke and gallinarum antigens and incomplete at 1:500 with pullorum antigen. The blood-serum of the tom failed to agglutinate any of the antigens. The blood-serums of three of the females did not contain any agglutinins for pullorum antigen at the time of slaughter.

FEEDING TESTS—ARTIFICIALLY INFECTED BIRDS

Three adult turkey hens (973, 975 and 977) were used in this experiment. These birds had been tested regularly by the test-tube method at monthly intervals from September, 1936, to April, 1937. Their blood-serums during this time were not found to contain any agglutinins with antigens used in this study. Each bird received, by way of a tube inserted into the crop, a saline suspension containing viable *S. aertrycke* organisms. The dates on which the birds were fed the suspension, the amount administered, and the concentration of the bacterial suspension are given in table I.

TABLE I—*Data on feeding tests of Salmonella aertrycke.*

DATE (1937)	DOSAGE (CC)	CONCENTRATION (%)
4- 8	6	1.375
4- 9	5	0.7
4-10	6	0.65
4-12	6	1.15
4-13	5	0.79

The rapid whole-blood plate agglutination test with pullorum and aertrycke antigens was made daily from April 8 to May 8, inclusive. Each day during this period fecal samples were collected, and blood-cultures were made. The blood was withdrawn from the wing vein with a sterile syringe and needle, and was immediately transferred to tubes containing 5 cc of plain broth. The cultures were incubated 72 hours and then plated on Endo agar plates. Colonies were transferred to serum-agar slants and then inoculated into carbohydrates. A Gram-positive coccus was obtained on five occasions. *S. aertrycke* was not recovered from the blood by this method.

FECAL EXAMINATIONS

Fecal samples were collected daily from April 8 to May 8. A portion of the fecal sample was transferred into infusion beef broth and incubated for about two hours. The broth cultures were then plated on eosin-methylene blue plates and Endo agar

TABLE II—*Recovery of Salmonella aertrycke from feces.*

DATE (1937)	BIRD
4- 9	973, 975
4-10	975
4-11	975, 977
4-12	973, 975
4-13	975, 977
4-14	973, 975
4-15	973, 975
4-16	977
4-17	973

plates. The plates were incubated for 24 hours and then transfers of colonies were made onto serum-agar slants. The cultures were incubated for 24 hours and then inoculated into carbohydrates. *S. aertrycke* was recovered from the fecal material of the birds on the dates as indicated in table II.

From April 19, 1937, to May 8, 1937, *E. coli communis* and *E. coli communior* were recovered consistently, and occasionally a staphylococcus.

AGGLUTINATION TEST

The whole-blood plate test was made daily during this period. A slight reaction appeared on the sixth day. The tube test on the seventh day showed that the serums contained agglutinins. Table III is a record of the test-tube agglutination tests of the three birds (973, 975 and 977) which were artificially fed viable *S. aertrycke* organisms as indicated before. A list of the antigens used, the dates when the tests were made, and the results of the tests are included.

The maximum titre was reached on the 13th day, and began to recede after the 21st day.

The birds were destroyed on September 27, and *S. aertrycke* was not recovered by bacteriological examination of the liver, spleen, heart, kidney, ovary, oviduct, bone-marrow, and intestinal contents.

Eggs were collected from the three birds (977, 975 and 973) from April 1 to July 22, 1937.

Eighty-seven eggs were incubated. Of this number, 50 were infertile, 14 had dead embryos at the end of the second week of incubation, six more had dead embryos at the end of the third week, and eleven additional dead embryos were found in the shells at the end of incubation. Five live

TABLE III—*Record of test-tube agglutination tests of birds 973,975 and 977.*

BIRD	ANTIGEN USED	DATE OF TEST			
		4-14-37	4-21-37	5-3-37	9-27-37
973	*S. aertrycke*	++++++	++++++	+++----	------
	S. pullorum	++I----	++I----	++I----	+I----
	S. equi	------	------	------	------
	S. suipestifer	------	------	------	------
	S. paratyphi A	------	II----	------	------
	S. paratyphi B	+II----	++++I-	++I----	+I----
	S. enteritidis	II----	II----	++----	------
	S. gallinarum	+I----	++I----	III----	------
975	*S. aertrycke*	+++----	++++I-	III----	------
	S. pullorum	++I----	++++--	III----	------
	S. equi	-------	I------	-------	------
	S. suipestifer	------	------	------	------
	S. paratyphi A	++----	++----	II----	------
	S. paratyphi B	+++----	++++++	III----	------
	S. enteritidis	------	II----	+++I--	------
	S. gallinarum	+++----	++II--	II----	------
977	*S. aertrycke*	+++----	++++--	++----	------
	S. pullorum	I------	++I----	------	------
	S. abortus-equi	------	------	------	------
	S. suipestifer	------	------	------	------
	S. paratyphi A	------	------	------	------
	S. paratyphi B	+++----	++++--	+++----	------
	S. enteritidis	------	------	------	------
	S. gallinarum	I------	I------	------	------

poults were hatched, of which only three survived and one died while breaking the shell.

It will have been observed that many of the above eggs were found infertile; others contained dead embryos when the eggs were tested at weekly intervals. The eggs were examined bacteriologically. *S. aertrycke* was not isolated. *E. coli* was isolated from nine eggs, staphylococci from eight eggs, Gram-positive bacilli from three eggs, and 61 eggs were sterile.

SYMPTOMS

The affected poults usually appeared normal until a short time before death, at which time some of the birds showed ruffled feathers, drooping wings, no desire to move, inappetence, and a thin, watery, foamy diarrhea. The region of the vent often was soiled with fecal material. They also acted as if they were chilled and would huddle under the hover, holding the head under the wing or on the breast.

POSTMORTEM FINDINGS

Autopsy findings were not definite. Some birds had an accumulation of serous fluid in the pericardial sac and abdominal cavity. The intestinal mucosa in some cases appeared slightly reddened and the lumen contained a dark, viscid, mucous exudate. The gizzard usually contained very little feed. Occasionally the ceca contained a hard, coagulated, cheese-like material.

CHARACTERISTICS OF SALMONELLA AERTRYCKE AND ASSOCIATED STRAINS

Serological: The Salmonella organisms isolated from the poults, eggs, and adult birds were sent to Dr. P. R. Edwards[2] for serological examination. The organisms isolated from the eggs and adult birds were serologically identical with *S. aertrycke*. The organism isolated from a 16-day-old poult was identified as serologically identical with *S. montevideo* of Hormaeche, and the organisms recovered from two eight-day-old poults and one seven-day-old poult were identified as *S. newington*.

Edwards[2] also examined for us, serologically, Salmonella organisms which were isolated from poults submitted to the Diagnosis Laboratory during the past two years. This group of cultures were obtained from 15 different farms.

One farm experienced trouble in 1935 and again in 1936. The organisms isolated in 1935 were serologically identical with *S. derby*. No birds were retained for breeding stock. In 1936, *S. aertrycke* was isolated from two different hatches two weeks apart. In 1937, the breeding flock was tested for pullorum and aertrycke infections and reactors removed. The owner encountered no trouble with the baby poults in 1937.

From another turkey farm, in 1937, Salmonella organisms were isolated from six different groups of baby poults. The poults were purchased from three separate hatcheries. The birds were maintained on two widely separated farms. Abnormal losses were encountered in certain hatches obtained from the three hatcheries. Edwards[2] again identified the organisms as follows:

(1) *Salmonella derby* was isolated in April from ten-day-old poults that originated in hatchery 1, farm 1.

(2) *Salmonella anatum* was isolated in April from 14-day-old poults that originated in hatchery 1, farm 1.

(3) A Salmonella (not identified) was isolated in May from ten-day-old poults that originated in hatchery 2, farm 2. This organism is a distinct type closely related to *Salmonella poona* and *Salmonella panama*.

(4) *Salmonella bareilly* was isolated in June from ten-day-old poults that originated in hatchery 3, farm 1.

(5) Salmonella rough strain belonging to the para B-aertrycke group was isolated in May from 14-day-old poults that originated in hatchery 2, farm 1.

(6) *Salmonella derby* was isolated in April from 14-day-old poults that originated in hatchery 1, farm 2.

One culture was found to belong to the b-enx series, having an undescribed somatic antigen. The organism is a distinct serologic type closely related to *S. paratyphi B* and *S. abortus-equi*.

The results of the serological examination of the other organisms which originated on the twelve remaining farms may be summarized as follows:

Salmonella aertrycke—7 farms
Salmonella derby—1 farm
Salmonella anatum—1 farm
Salmonella bredeney—2 farms
Salmonella senftenberg—1 farm

Morphological and cultural characteristics: *S. aertrycke* and associated strains are Gram-negative, motile, non-sporulating rods. Indol is not formed and nitrates are reduced to nitrites. The organisms grow readily on ordinary culture media.

The *S. aertrycke* strains of turkey origin which were studied formed acid and gas in the following carbohydrates: dextrose, levulose, galactose, arabinose, xylose, maltose, mannite, sorbitol, isodulcitol, inositol, dulcitol, mannose and trehalose. Acid was formed very slowly in glycerol. Lactose, sucrose, inulin, salicin, dextrin, raffiinose and adonitol were not affected by these strains. Litmus milk was slightly acidified and, after incubation for 48 hours to 72 hours, became alkaline. Stab cultures in lead acetate media produced no discoloration of the media. Jordan's media became yellow at the end of 24 hours of incubation, indicating acid formation. Later the media became pink again. Acid and gas were formed in the bottom of the tube containing Russell's double-sugar media; no change occurred on the slant. Cystine gelatin became cloudy along the line of inoculation.

The *S. newington* strains varied from *S. aertrycke* strains in not fermenting xylose, glycerol and inositol. In Russell's double-sugar media *S. newington* produced acid on the slant and acid and gas in the bottom of the tube.

The *S. montevideo* strain failed to attack glycerol and inositol and was slow in attacking dulcitol.

The *S. bareilly* strain failed to attack glycerol. Acid was formed on the slant and in the bottom of the tube containing Russell's media.

The *S. anatum* strains produced acid and gas in glycerol and failed to attack inositol.

The *S. derby* strains agreed with the carbohydrate reactions of *S. aertrycke*.

The *S. senftenberg* strain failed to ferment inositol but produced acid and gas in glycerol.

During the past ten years, considerable research work has been done on incubator hygiene in regard to *S. pullorum* transmission in baby chicks. The experimental work has resulted in standard recommendations for incubator hygiene. Additional research work is necessary to determine methods of control of diseases of poults resulting from *S. aertrycke* and associated organisms. The application of agglutination tests for the diagnosis of pullorum and aertrycke infection in adult turkeys requires further study.

SUMMARY

1. *Salmonella aertrycke* may produce an acute disease of young poults up to five weeks of age.

2. *S. aertrycke* was isolated from infertile eggs and dead-in-the-shell embryos during the second, third and fourth week of incubation.

3. *S. aertrycke* was isolated from the ovary and oviduct of three of ten naturally infected birds.

4. In addition to *S. aertrycke*, *S. anatum*, *S. newington*, *S. montevideo*, *S. derby*, *S. senftenberg*, *S. bareilly* and *S. bredeney* may produce losses among baby poults.

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DISCUSSION

DR. HUGH HURST: I should like to know if it would make any material difference in the welfare or final mortality of the flock if these birds were affected with *Salmonella pullorum*.

DR. POMEROY: It would vary in some flocks. I would consider paratyphoid infection as serious a problem as pullorum disease.

DR. M. W. EMMEL: I think this question of paratyphoid in turkeys is one of the very important problems that we have. In Florida we never like to see it, because it just wipes out the entire flock from eight weeks on down. There seems very little we can do about it.

I would like to ask Dr. Pomeroy if, in these affected flocks, any record of egg production was kept, or does he have any record of egg production in affected flocks as compared with those not affected.

DR. POMEROY: No, we do not. Most of the original infections were traced to eggs imported into the state, and we had no information as to the breeding stock.

DR. HURST: Is the pullorum test thoroughly dependable to detect paratyphoid?

DR. POMEROY: I do not know. Cross-agglutination between *Salmonella aertrycke* and *Salmonella pullorum* does occur, and the pullorum test would detect many of the aertrycke-infected birds.

DR. EMMEL: Do you believe that the infection in the hen is carried over from infection as a poult, or does the hen become infected later in life?

DR. POMEROY: I believe it is carried over from infected poults.

DR. EMMEL: I asked that because it substantiated what you said a while ago about pullorum and aertrycke. We have been doing work with paratyphoid infections, particularly in turkeys, and even in adult turkeys we find the suspension of aertrycke that we can inject intravenously is very, very small as compared to any other paratyphoid organism we can inject. The turkey is much more susceptible to paratyphoid than any other organism.

Proved Sires Boost Dairy Herd Output

Production records are kept on only a little more than 2 per cent of the milk cows on American farms, reports Dr. J. F. Kendrick, in charge of dairy herd-improvement work for the Bureau of Dairy Industry. Of the more than 25,000,000 milk cows in the country, only about 560,000 are on test in the 1,100 dairy testing associations that have been organized by state dairy extension agencies in their respective states.

This small proportion, however, exerts a tremendous influence on milk production, not only within the herds on test but also on herds that have never been tested and possibly never will.

From this 2 per cent, Dr. Kendrick points out, dairy sires are now being proved at the rate of about 1,000 head per year. When production records are more complete—the nation-wide sire-proving work has been under way for only two years—from 2,000 to 3,000 sires are expected to be proved annually.

The sires that show up well are the "blue bloods" of the dairy cattle world. Although the large majority of dairy farmers do not keep records on their own cows, they do know the value of a good proved sire. By bull associations, artificial insemination, and other improved practices, farmers use to advantage work that is being done by the few dairymen who keep records.

Game Supply in Forests Governs Hunting Rules

Supply rather than demand controls Forest Service policy in encouraging or limiting hunting, trapping and fishing in the national forests, says Dr. H. L. Shantz, chief of the Division of Wildlife Management of the federal Forest Service. Under this policy of conserving game, several species of wildlife have increased until the supply warrants encouragement of hunting.

For every three deer in the national forests in 1924, there are now eight. There is now good deer hunting in most of the western forests, in the Lake States, and in Pennsylvania, North Carolina and Florida. The Forest Service believes that first call on the deer surplus should be used in restocking areas where deer have been killed out. After this, says Dr. Shantz, true conservation demands hunting enough to control the surplus and keep the number of deer within the feeding capacity of the range.

Another Forest Service aim is to maintain a rich plant cover as well as a varied and interesting population of wildlife. An oversupply of deer would soon injure the plant cover. By adjusting the number of hunting permits and the bag limits, with the coöperation of state game officials, the Service is finding it practical to fit the hunting to the supply.

An Alum-Precipitated Toxoid as an Immunizing Agent Against Infectious Necrotic Hepatitis (Black Disease) in Sheep*

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Infectious necrotic hepatitis is an acute infectious disease of sheep and, occasionally, of cattle, caused by the localized activity of *Clostridium edematiens* in necrotic foci in the liver following invasion by the immature liver fluke, *Fasciola hepatica*. Death is produced by toxemia caused by the rapid elaboration of toxin by *C. edematiens*. The common name of this disease is "black disease," which is descriptive of the large, dark red, subcutaneous hemorrhages found on the pelt after removal from the sheep.

This disease has been known clinically for at least 60 years in Australia, where the name "black disease" originated (Turner[1]). It was reported in New Zealand by Hopkirk.[2] Bull and Turner[3] state that it has been found in Germany, France, Rumania and Chile. There may be some confusion between this disease and the disease known as "braxy" in the European literature, in which the causative organism has been generally accepted to be *C. edematis*. The literature contained a great deal of valuable information on black disease at the time Turner[1] published his monograph, in 1930. It is the most complete work to be found on the subject.

Included in Turner's monograph is a bibliography in which he cites the bacteriological investigations made by Gilruth (1910, 1911), Dodd (1918, 1921), Hopkirk (1927), Albiston (1927), Turner and Davesne (1927), and Edgar (1929). All of these investigators isolated the causative organism from necrotic areas in the liver. Dodd did not recognize the organism as *C. edematiens;* Turner and Davesne identified the organism isolated by Albiston in Victoria as *C. edematiens;* and Edgar identified the causative organism of the disease in New South Wales as *C. edematiens*. Edgar demonstrated the presence of *C. edematiens* in livers of normal sheep from infected premises and the absence of the organism in livers of sheep from localities which were free from black disease losses.

Dodd and subsequent investigators noted the constant association of fluke infestation with black disease, and Turner experimentally proved the relation of the flukes to the development of the disease. Turner confirmed Edgar's work in demonstrating spores of *C. edematiens* in the livers of normal sheep pastured on infected premises, and he found the same organism in livers of apparently normal rabbits captured on infected areas. A fatal infection in rabbits, resembling black disease, was produced by using toxin-free spores with calcium ions as a tissue irritant. The infection was reproduced in rabbits by feeding eggs of *Taenia serrata*, followed by the administration of spores. The typical picture was reproduced by feeding laboratory-reared cercariae of *F. hepatica* to guinea pigs, with subsequent injections of toxin-free spores.

There have been no published reports of the existence of this disease in the United States. In 1925, Huffman and Dade[4] reported losses of sheep in Idaho from a disease associated with the presence of liver flukes, which was probably black disease. Shaw and Simms,[5] in discussing liver-fluke disease in 1930, refer to sudden deaths in sheep when only a few flukes are present, but state that they had been unable to prove that pathogenic anaerobes were responsible. In another publication, the following year, Shaw[6] reported that it "seems

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†Dr. W. J. Kilpatrick, of Hamilton, Mont., worked with the authors throughout the period covered by this investigation, obtaining material from field cases and carrying out the field immunization work.
‡Montana Experiment Station and Montana Livestock Sanitary Board coöperating.

to be definitely proven by the British" that secondary invaders are responsible for such sudden deaths, but that "so far in this country secondary invading organisms have not been incriminated experimentally."

In 1930, investigations were started in Montana to determine the cause of death losses in the farm flocks of sheep in the Bitterroot Valley. These investigations resulted in a diagnosis of black disease, based on the clinical history and pathological lesions, and the finding of *C. edematiens* in the necrotic areas of the livers, with the presence of flukes (Marsh[2]).

OCCURRENCE IN MONTANA

The disease as observed in Montana has occurred in farm flocks on the irrigated ranches in the Bitterroot Valley, which is west of the continental divide. This valley is known to be heavily infested with liver flukes, both in sheep and in cattle. Fluke disease has been recognized in many of the farm flocks of sheep for a number of years, and some flocks have been repeatedly treated for liver flukes. The losses which we have diagnosed as black disease have occurred in flocks where the effects of liver-fluke infestation have not been apparent.

The sheep affected have been mature ewes in excellent condition. No reports of losses in lambs have been received. The occurrence of the disease is seasonal, all cases reported having occurred between August 1 and April 1. The deaths in any one affected flock are distributed through the fall and winter. One owner reported a loss of 75 from a flock of 300 ewes during the fall and winter of 1930-1931, no more than two dying on any one day. A number of owners have lost sheep in this proportion, with the result that, within the past two years, many owners have gone out of the sheep business.

SYMPTOMS

Black disease has a mortality of 100 per cent, and, as observed on the ranches, death is almost the only symptom. Usually the sheep are found dead on the bed-ground in the morning, without having shown any symptoms of sickness. Occasionally a ewe is noticed as slightly depressed, and the next day she is dead.

PATHOLOGY

Because postmortem change takes place quite rapidly, the number of satisfactory autopsies is rather small. The lesions are very characteristic of this disease. One of the prominent lesions is the subcutaneous hemorrhage on the back and sides, which gives a dark color to the flesh side of the pelt and is responsible for the name "black disease." Another very characteristic lesion is the distention of the pericardium with clear fluid, and petechiae or ecchymoses on the endocardium. The thoracic and abdominal cavities usually contain an excess of fluid.

On casual inspection, the liver often appears quite normal, but close examination always reveals some changes. There are dark hemorrhagic areas and several typically necrotic foci. These foci are rather ill-defined, and in some cases the only gross change is a softening of the tissue in the area. Beneath the capsule there are usually visible the scars of the tracks of immature flukes which have migrated from the surface of the liver to the bile-ducts. A few flukes are usually to be found in the liver, although in several livers which have been sent to the laboratory no flukes were found. In no case observed has there been extensive fluke infestation of the liver.

The kidneys may show irregular necrotic areas 5 to 8 mm in diameter similar to those found on the liver. The pyloric region of the abomasum has quite regularly presented an altered appearance. The mucosa shows varying degrees of congestion, from slight to severe, and a clear, gelatinous edema may be found beneath the mucous membrane. The intestinal mucosa shows patchy congestion. The omental fat may be congested, or even hemorrhagic.

The histopathological changes in the liver are characteristic. Sections through the gross necrotic areas show lesions which correspond to those described by Turner.[1] The characteristic lesion is a nodule whose center consists of cell débris, surrounded by a dense zone of polymorphonuclear leukocytes, lymphocytes and cells with distorted nuclei. Surrounding this dense zone is a zone of giant-cells, outside of which are large numbers of eosinophiles. There is also

infiltration of eosinophiles around portal veins and bile-ducts. In some cases the nodules are embedded in connective tissue, in which there is proliferation of bile-ducts.

Sections through areas where there is no gross change show degeneration of liver cells, with a catarrhal condition of the mucosa of the bile-ducts and cellular infiltration around the ducts. There are small necrotic foci near the portal veins, with masses of large Gram-positive bacilli at the centers. Gram-positive bacilli are also scattered through the liver tissue.

BACTERIOLOGICAL FINDINGS

Bacteriological examination of tissues from cases of black disease has not been satisfactory except under the most favorable conditions. The tissues must be removed from the sheep immediately after death and must be held at a low temperature until the cultures are made. Under these conditions it has been possible quite regularly to recover *C. edematiens* from the hemorrhagic and necrotic areas in the liver. This organism has also been recovered from the wall of the abomasum, the kidney, and skeletal muscle.

IMMUNIZATION

Since 1921, various immunizing products have been prepared against black disease by workers in the Southern Hemisphere. The most extensive work along this line was done by Turner. He prepared a toxoid which produced a limited immunity in vaccinated rabbits. His next immunizing product was an anaculture. Guinea pigs were found to be very unsatisfactory in their immunity response. Sheep thrice vaccinated gave a satisfactory immunity response when exposed to as much as 20 mld of culture. The once-vaccinated group resisted 2 mld of the culture. Turner's[8] field vaccination trials with the anaculture over the period 1928-1934 show that 13,742 unvaccinated controls suffered a death loss of 3.65 per cent; whereas, in 16,337 twice-vaccinated sheep there was a loss of 1.34 per cent, while in the 4,888 thrice-vaccinated sheep only .49 per cent were lost.

In 1937, Oxer[9] published the results of his work with an alum-precipitated anaculture of *C. edematiens*, one dose of which produced a much higher degree of immunity in guinea pigs than two doses of the Turner anaculture. His experimental tests of the immunity produced by the alum-precipitated vaccine in sheep were unsatisfactory, because the method of artificial exposure of the sheep was not entirely successful.

The work reported here was carried on simultaneously with Oxer's investigations, but was not completed until after his publication appeared. As several phases of our work covered points which appear to add to the useful information on this subject, it is considered worth while to report our results. In 1931 and 1932, preliminary work was done in which the methods described by Turner were used in an attempt to produce an effective anaculture. These preliminary experiments did not produce satisfactory results. It was found that some strains of *C. edematiens* isolated from cases of black disease failed to produce a strong toxin in cultures, and that artificial cultivation decreased the pathogenicity and capacity for toxin production. Another handicap was the difficulty of recovering suitable strains from livers shipped to the laboratory.

In the fall of 1935, the facilities of the Rocky Mountain Laboratory of the United States Public Health Service, located at Hamilton, Montana, in the area where the black disease losses were occurring, were kindly placed at our disposal. It was thus possible to make bacteriological examination of tissues immediately after death. As a result, a highly toxic strain of *C. edematiens* was isolated, and several toxic strains have been isolated at intervals during the past two years.

With freshly isolated, highly toxic cultures available, anacultures were prepared. The medium used was the "VF" peptic digest broth, prepared as described by Turner, with the addition of 0.5 per cent dextrose. To this medium there was also added a number of 4- to 6-inch lengths of iron baling wire (10 per cent by weight), which, controlled experiments showed, increased the growth of this organism, presumably by acting as a reducing agent. After growth for 48 hours in this medium, 1 per cent for-

malin was added to kill the organisms and change the toxin to toxoid. It was found that exposure for three days to 1 per cent formalin produced sterility and detoxification of the anaculture.

Guinea pigs were vaccinated with the anacultures and the immunity response measured. Results typical of all these anacultures are given in table I, in which anaculture 989 was used as the immunizing agent. After detoxification and sterility were assured, guinea pigs weighing 300 to 400 gm were twice vaccinated, five days apart, with 0.5 cc of anaculture 989. The exposure was made 36 days after the second vaccination, using an intramuscular injection of a whole culture of *C. edematiens* (strain 989). The culture was grown 51 hours in Haslam's[10] brain-liver medium, containing iron wire. The vaccinated pigs were exposed with 1, 2, and 3 mld of culture. Protection was complete in the vaccinated pigs against 1 and 2 mld, but two of three pigs receiving 3 mld died. The three controls receiving 1 mld died.

TABLE I—*Immunization of guinea pigs with two 0.5-cc doses of anaculture.*

G. P.	Vaccination		Exposure*			Result
	First	Second	Date	Material	Dose	
1	4–25	4–30	6–5	Whole culture	1.0 mld	Lived
2					1.0 mld	Lived
3					1.0 mld	Lived
4					2.0 mld	Lived
5					2.0 mld	Lived
6					2.0 mld	Lived
7					3.0 mld	Lived
8					3.0 mld	Died
9					3.0 mld	Died
10	Controls				0.5 mld	Lived
11					0.5 mld	Died
12					1.0 mld	Died
13					1.0 mld	Died
14					1.0 mld	Died

*Inoculated intramuscularly.

Having demonstrated that a double vaccination with an anaculture produced immunity in guinea pigs against 2 mld of the culture, an experiment was set up to determine whether immunity could be produced in sheep. A group of ewes was twice vaccinated, nine days apart, with 5 cc of anaculture 989. Exposures by intramuscular inoculation of culture 989 were made 28 days after the second vaccination. The culture was grown 50 hours in brain-liver medium. A preliminary titration of the culture on three ewes placed the mld at 0.5 cc. The toxicity of the culture was checked both in the preliminary test and in the final exposure by guinea-pig titration. Table II shows the results of the final controlled test on the vaccinated sheep. Taking 0.5 cc as the mld for sheep, it will be seen that, of the six vaccinated sheep receiving 1 to 4 mld of culture, only three survived. The control sheep receiving 1 mld died. These results indicate the production of some immunity, but the protection was not sufficient to be of any practical value.

CONCENTRATED ANACULTURE

In seeking an explanation for the fact that a solid, though limited, immunity was produced by the anaculture in guinea pigs but not in sheep, it seemed possible that the difference in the amount of immunizing agent in proportion to the weight of the animal might have been the factor. The dose of anaculture used for the sheep was only ten times as great as the dose for the guinea pig, while the weight of the sheep was about 150 times as great as the weight of the guinea pig.

Accordingly, an anaculture was prepared which will be designated as a "concentrated anaculture," in which part of the culture fluid was discarded, thus concentrating the bacterial bodies. This anaculture was prepared by the usual method of formalinizing the whole culture from a two-day growth of

TABLE II—*Immunization of sheep with two 5-cc doses of anaculture.*

EWE	VACCINATION		EXPOSURE*				RESULT
	FIRST	SECOND	DATE	MATERIAL	DOSE		
					CC	MLD	
1	6–9	6–18	7–16	Whole culture	0.5	1.0	Lived
2					0.5	1.0	Died
3					0.5	1.0	Lived
4					1.0	2.0	Died
5					1.0	2.0	Lived
6					2.0	4.0	Died
7	Controls				0.25	0.5	Lived
8					0.5	1.0	Died

*Inoculated intramuscularly.

C. edematiens 989 in peptic digest broth, pH 7.4, plus 0.5 per cent dextrose and iron wire. The bacterial cells from 4,000 cc of the anaculture were concentrated by centrifugation and resuspended in the supernatant fluid to a volume of 280 cc. This suspension constituted the so-called "concentrated anaculture" used for the immunization tests with sheep and guinea pigs.

A group of guinea pigs and two groups of sheep were twice vaccinated, five days apart, using an intramuscular injection of the "concentrated anaculture." The pigs received 0.5 cc; one group of ewes, 5 cc; and the other group of ewes, 10 cc, at each inoculation. The original potency of the toxin from this anaculture before formalinization was such that 0.00,009 cc per gram of guinea pig weight constituted 1 mld for guinea pigs. Exposure of the vaccinated guinea pigs was made 4½ months after the second injection, by intramuscular inoculation with a 43-hour culture grown in brain-liver medium. A very uneven immunity response was obtained, in that only two of seven pigs had sufficient immunity to withstand 10 to 40 mld of culture. Two of the controls receiving 1 mld died (table III).

The vaccinated sheep were exposed five months after the second immunizing inoculation, by using an intramuscular injection of 48-hour cultures grown in brain-liver medium. A preliminary titration of the culture in sheep proved 0.001 cc to be the equivalent of at least 1 mld. Among the vaccinated sheep, only five of the nine survived an exposure of 2½ to 5 mld. All three controls receiving 2½-mld inoculations died. The larger dose of vaccine (10 cc) produced no more immunity than did the smaller quantity (table IV). Such unreliable immunity response, while somewhat better than that produced by the previous anacultures, would hardly justify the use of the concentrated anaculture in the control of the disease.

TABLE III—*Immunization of guinea pigs with two 0.5-cc doses of concentrated anaculture.*

G. P.	VACCINATION		EXPOSURE*			RESULT
	FIRST	SECOND	DATE	MATERIAL	DOSE	
1	7–29	8–3	12–23	Whole culture	10 mld	Died
2					20 mld	Died
3					20 mld	Lived
4					20 mld	Died
5					40 mld	Died
6					40 mld	Lived
7					40 mld	Died
8					60 mld	Died
9	Controls				1 mld	Died
10					1 mld	Died
11					1 mld	Lived

*Inoculated intramuscularly.

COMPARISON OF ANACULTURE, TOXOID AND BACTERIN

Experiments with immunizing agents up to this time had been based on the theory of the production of bactericidal antibodies, as preliminary tests of the toxoid had produced unsatisfactory results. As the concentration of bacterial bodies failed to increase materially the immunizing value of the anaculture, an experiment was set up to compare the immunizing value of the whole anaculture, the bacterial bodies alone, and the culture filtrate (toxoid) alone. For this experiment there was available a strain of *C. edematiens* of much higher toxin-producing power than the strain previously used.

This strain (1146) was recently isolated from a typical case of black disease in a sheep. The culture was grown for 48 hours in peptic digest broth, pH 7.4, containing 0.5 per cent dextrose and 10 per cent iron wire. After detoxification and sterilization with 1 per cent formalin, a portion of the anaculture was centrifuged. The supernatant fluid was then passed through a Mandler candle, for use as a toxoid. The packed bacteria were washed several times and finally resuspended to the original volume in sterile peptic digest broth, to be used as the bacterin. The anaculture, consisting of toxoid and formalin-killed bacteria, was used in its original form. Before formalinization of the toxin, the guinea pig mld was established at 0.000,009 cc per gram of body weight.

Three groups of guinea pigs were twice vaccinated, five days apart, by intramuscular injections with 0.5 cc of the anaculture, the toxoid, and the bacterin, respectively. These vaccinated pigs were exposed, 32 to 37 days after the second vaccination, by intramuscular injections of toxic 48-hour cultures grown in brain-liver medium. The pigs immunized with the toxoid and the anaculture withstood inoculations of 100 mld, but the bacterin-vaccinated group was unable to survive 5 mld of the same inoculum (table V). These results proved quite conclusively that the immunizing value of the anaculture had been derived from the toxoid rather than the bacterial cells.

TABLE IV—*Immunization of sheep with two doses of concentrated anaculture.*

Ewe	Vaccination				Exposure*				Result
	First		Second		Date	Material	Dose		
	Date	Dose (cc)	Date	Dose (cc)			cc	mld	
1		5.0		5.0	1-9		.0025	2.5	Lived
2		5.0		5.0	1-9		.0025	2.5	Died
3		5.0		5.0	1-9		.005	5.0	Lived
4		5.0		5.0	1-9		.005	5.0	Lived
5	7-29	10.0	8-3	10.0	1-4	Whole	.0025	2.5	Died
6		10.0		10.0	1-4	culture	.0025	2.5	Died
7		10.0		10.0	1-4		.0025	2.5	Lived
8		10.0		10.0	1-4		.0025	2.5	Lived
9		10.0		10.0	1-9		.005	5.0	Died
10					1-4		.0025	2.5	Died
11		Controls			1-4		.0025	2.5	Died
12					1-9		.0025	2.5	Died

*Inoculated intramuscularly.

PRECIPITATED TOXOID

The literature shows that alum precipitation increases the immunizing efficiency of a toxoid. The toxoid to be discussed was prepared by applying the results of the work of Havens and Wells[11] and Smith.[12] The medium used for toxin production was the same as that employed for the anacultures; namely, peptic digest broth, pH 7.2-7.4, to which was added 0.5 per cent dextrose and 10 per cent iron wire. A 52-hour growth, at 37° C., of *C. edematiens* (strain 1146) was used. The mld of the filtrate was 0.000,005 cc per gm of weight for guinea pigs. Subsequent tests of other

batches of toxin produced from this strain showed about the same unitage of toxin at 40 to 48 hours.

The 52-hour culture was formalinized with 1 per cent formalin, the flasks tightly stoppered, and incubated at 37° C. for three days, until completely detoxified and sterilized. The toxoid was then recovered by filtration through cotton, moist paper pulp, and a Mandler candle. The toxoid was precipitated by adding 1.5 per cent potassium alum from a sterile 10 per cent solution. The alum solution was sterilized by filtration through a Mandler candle. Mercurophen solution was added as a preservative, to a concentration of 1:10,000.

In determining the concentration of alum which would yield the maximum precipitate, a preliminary test was made, using 1, 1.5, 2, and 2.5 per cent concentrations of alum. A 35 per cent yield of precipitate was obtained with the 1.5 per cent concentration after standing 11 hours; whereas, the 2 per cent and 1 per cent concentrations produced 28 per cent and 30 per cent, respectively. Oxer found that 2 per cent was the optimum amount of alum for precipitation of the anaculture.

The experimental toxoids reported upon in this paper were prepared as described. However, when mass production was considered, it became advisable to institute certain modifications in the technic. The first change to be effected was in the preparation of the "VF" broth. Rather than employ the fresh pig stomachs as a source of the enzyme, a commercially-prepared trypsin was substituted, with equally good results. Under certain conditions, some toxin is lost through adsorption by the filter candles. Moreover, the use of filter candles adds an unnecessarily costly step to the manufactured product. Both objections have been overcome by removing the bacteria by high-speed centrifugation instead of filtration.

TABLE V—*Comparison of immunizing value of anaculture, toxoid and bacterin for guinea pigs.*

G. P.	Immunizing Agent	Vaccination		Exposure*			Result
		First	Second	Date	Material	Dose (MLD)	
1	Anaculture	2.1	2.6	3-10	Whole culture	2.0	Lived
2						3.0	Lived
3						5.0	Lived
4						5.0	Lived
5						6.0	Lived
6						10.0	Lived
7				3-15		50.0	Lived
8						100.0	Lived
9	Toxoid			3-10		2.0	Lived
10						3.0	Lived
11						5.0	Lived
12						5.0	Lived
13						6.0	Lived
14				3-15		10.0	Lived
15						10.0	Lived
16						50.0	Lived
17						100.0	Lived
18	Bacterin			3-10		2.0	Lived
19						3.0	Lived
20						5.0	Lived
21						5.0	Died
22						6.0	Died
23	Controls			3-10		1.0	Died
24						1.0	Died
25						1.0	Lived
26				3-15		1.0	Died
27						1.0	Lived
28						1.0	Lived

*Inoculated intramuscularly.

IMMUNIZATION WITH PRECIPITATED TOXOID

A group of guinea pigs weighing 400 to 650 gm and a group of mature ewes weighing 100 to 150 pounds were vaccinated with the alum-precipitated toxoid. The guinea pigs were given two 0.5-cc subcutaneous injections, eight days apart, and the sheep received one 5-cc inoculation subcutaneously.

TABLE VI—*Immunization of guinea pigs with alum-precipitated toxoid.*

G. P.	VACCINATION		EXPOSURE*			RESULT
	FIRST	SECOND	DATE	MATERIAL	DOSE (MLD)	
1	5–14	5–22	6–14	Toxin (culture filtrate)	10.0	Lived
2					25.0	Lived
3					50.0	Lived
4					75.0	Lived
5					100.0	Lived
6			6–18		200.0	Lived
7					300.0	Lived
8					400.0	Lived
9	Controls		6–14		1.0	Died
10					1.0	Died
11					1.0	Died
12					1.0	Lived
13					1.0	Died
14					1.0	Died
15			6–18		1.0	Died
16					1.0	Lived
17					1.0	Died
18	5–14	5–22	10–8	Precipitated toxin (lot 1)	166.0	Lived
19					166.0	Lived
20					250.0	Lived
21					250.0	Lived
22					333.0	Died
23	Controls		10–8		1.0	Died
24					1.0	Died
25					1.0	Lived
26					1.0	Lived

*Inoculated subcutaneously.

In testing the immunity produced by the alum-precipitated toxoid, the method of exposure was changed from that used in previous immunity tests. In place of the whole culture inoculation, an injection of toxin was used. In the case of guinea pigs, this change made no difference in the results, as it was found that the injection of toxin produced the same result in guinea pigs as did the inoculation with the whole culture. However, with sheep this change was of great importance.

Intramuscular injection of whole cultures produced a characteristic local edema, followed by death after several days. This condition was not comparable to black disease as it occurs in the field. In black disease the invasion of tissue is slight, but the toxemia is fatal. It was found that the intravenous injection of toxin, as compared with the intramuscular inoculation of culture, resulted in the production of typical black disease as observed in the field, with the characteristic pathological lesions, except the liver changes.

Twenty-three days after the second vaccination, five of the guinea pigs were exposed to the toxin (culture filtrate) of the homologous strain (1146) of *C. edematiens* by the subcutaneous injection of 10 to 100 mld. Twenty-seven days after the second vaccination, three more pigs were exposed to 200 to 400 mld of the same toxin. The potency of the toxin used for each exposure was such that it required 0.00,003 cc per gm of body weight to kill the control guinea pigs. At the first exposures, five of six controls given 0.00,003 cc died, and on the sec-

ond test two out of three controls given 0.00,003 cc succumbed. All of the eight immunized pigs remained well. These results proved that a strong immunity had been produced, which lasted for at least 27 days.

Some of the same group of immunized pigs were carried 139 days for a test on the duration of immunity. For this test a precipitated toxin was used, the mld of which was 0.0,006 mg per gm of guinea-pig weight. Two vaccinated pigs survived the subcutaneous inoculation of 250 mld of toxin, but the one exposed to 333 mld died. These results indicate that the initial immunity, while still strong, had dropped somewhat during the 112-day interval between tests. The data on guinea-pig immunization are shown in table VI.

The results of the immunization of the sheep with precipitated toxoid are shown in table VII. Six immunized and three control sheep were exposed by intravenous injection of culture filtrate 42 to 47 days following vaccination. The results with the control sheep show that the mld was approximately 2 cc. The vaccinated sheep were completely protected against a dosage of 12 mld, which was the largest quantity used.

The rest of the immunized sheep were continued on the experiment to test the duration of immunity. On October 1, 140 days after immunization, three sheep exposed by the intravenous inoculation of 4 mld of precipitated toxin (lot 1) proved immune. By a preliminary titration of lot 1 toxin with two control sheep, the mld was determined to be 0.5 gm. Due to a shortage of toxin in lot 1, it became necessary to use another preparation (lot 2) made from the same strain, but with a greater toxin unitage. Lot 2 contained at least 1 sheep mld in 0.37 mg per pound of body weight (table VIII). On October

TABLE VII—*Single vaccination of sheep with 5-cc of alum-precipitated toxoid.*

Ewe	Vaccination	Exposure*				Result
		Date	Material	Dose		
				cc or gm	mld	
1	5-14-37	6-25-37	Toxin (culture filtrate)	6.0	3.0	Lived
2				6.0	3.0	Lived
3		6-28-37		20.0	10.0	Lived
4				20.0	10.0	Lived
5		6-30-37		20.0	10.0	Lived
6				25.0	12.5	Lived
7	Controls	6-21-37		1.0	—	Lived
8		6-25-37		2.0	1.0	Died
9		6-23-37		2.5	—	Died
10	5-14-37	10-1-37	Precipitated toxin (lot 1)	2.0	4.0	Lived
11				2.0	4.0	Lived
12				2.0	4.0	Lived
13	Controls	9-20-37		0.26	—	Lived
14		9-23-37		0.5	1.0	Died
15	5-14-37	10-7-37	Precipitated toxin (lot 2)†	0.2	5.0	Lived
16				0.2	5.0	Lived
17				0.2	5.0	Lived
18				0.8	15.0	Lived
19				0.8	16.0	Lived
20		1-14-38		0.2	4.0	Lived
21				0.4	8.0	Lived
22				0.8	18.0	Lived
23		1-22-38		0.91	22.0	Lived
24				0.93	22.0	Lived
25				1.88	34.0	Lived

*Inoculated intravenously.
†For control sheep on precipitated toxin, lot 2, see table VIII.

7, 146 days after vaccination, five ewes (15 to 19, table VII) were tested for immunity with lot 2 toxin. Three sheep received 5 mld; one, 15 mld, and one, 16 mld of toxin, and all survived the exposure.

As a further test upon the question of the duration of immunity, the remaining unexposed vaccinated sheep were continued on the experiment. Three of these ewes (20 to 22) were tested for immunity on January 14, and three more (23 to 25) on January 22, 1938, 245 and 251 days, respectively, after vaccination. All of these sheep were completely protected, the highest dose of toxin being 34 mld.

As the investigation proceeded, it became apparent that the quantity of toxin required to produce fatal results in the control sheep was directly proportional to the body weight, as was the case with guinea pigs. This fact is shown in table VIII, in which the mld was quite closely determined for lot 2 toxin. It will be seen that sheep 2 survived the injection of 0.3 mg of toxin for each pound of body weight, while sheep 3 died when injected with 0.37 mg per pound, which was accepted as the minimum lethal dose. These animals were all mature ewes in good physical condition, varying in weight from 99 to 150 pounds. These results established the mld for this lot of toxin, which was then used in the exposure of the vaccinated ewes during October and January. Control sheep were not exposed along with each group of vaccinated animals, because the potency of the toxin was determined to be constant by frequent guinea-pig titrations.

TABLE VIII—*Determination of minimum lethal dose of precipitated toxin (lot 2) for control sheep.*

Ewe	Weight (lbs.)	Dose		Result
		Total (gm)	Mg per Pound of Body Weight	
1	135	0.030	0.22	Lived
2	133	0.040	0.30	Lived
3	138	0.052	0.37	Died
4	119	0.049	0.41	Died
5	150	0.075	0.50	Died
6	99	0.050	0.50	Died

SHEEP IMMUNIZED BY AUSTRALIAN ANACULTURE AGAINST MONTANA TOXIN

To establish the immunological relationship between the disease as it occurs in Australia and in Montana, a group of sheep was immunized with the Australian vaccine and then exposed to toxin from a Montana strain. The vaccine was an alum-precipitated, concentrated anaculture, presumably prepared as described by Oxer. Mature ewes were immunized on May 14, by a single 2-cc subcutaneous inoculation with this anaculture. The immunity developed in these sheep was tested by the intravenous injection of the precipitated toxin (lot 2). One ewe was given 4 mld, and a second ewe, 5 mld, 146 days after vaccination; and a third sheep received 16 mld, 245 days after vaccination. These three sheep were completely protected, thus establishing the antigenic relationship between the strains of *C. edematiens* from Australia and Montana.

EXPERIMENTAL FIELD VACCINATION

After the laboratory experiments had demonstrated that an immunity could be produced by the precipitated toxoid which would protect sheep against an artificial exposure to *C. edematiens* toxin, a controlled field experiment was arranged to determine whether this immunity would protect against the natural infection. For this experiment six farm flocks, totalling 466 sheep, and one range outfit of 3,050 sheep, were used.

The farm flocks were selected on the basis of a known history of heavy losses from black disease over a period of years. The sheep in these flocks were vaccinated on July 13 and 14, giving time for the development of immunity before the period in which the first losses usually occur. The flocks on each ranch were divided so that approximately one-half of each flock was vaccinated, and the remainder were allowed to go unvaccinated.

In selecting the sheep for vaccination, the fat and thin ewes of each flock were sorted so that the vaccinated and control groups contained a representative number of each class. The numbers were small

enough so that each flock was run as a unit, thus giving all the animals equal opportunity for infection with the organism and infestation by the flukes. This procedure was true for the period before and after vaccination. Each vaccinated sheep received one 5-cc subcutaneous injection of the alum-precipitated toxoid.

Losses from black disease in these flocks were recorded for a period of seven months following vaccination, ending February 5, 1938, at which time all losses had ceased. Unfortunately, two of the flocks were disposed of by the owners before the test period was over, reducing the number to four, with a total of 344 sheep. The results from the four flocks are listed in table IX. The deaths were checked by a competent veterinarian familiar with this disease and, in so far as it was possible to determine, the deaths here listed resulted from black disease.

On ranch 1, no deaths occurred among the 24 immunized ewes, while two (8.7 per cent) of the 23 control ewes died. Among the sheep on ranch 2, none of the 33 immunized ewes died, while one (3.1 per cent) of the 32 controls died. On ranch 3, no losses occurred among the 64 immunized ewes, but four (6.2 per cent) of the controls died.

As previously stated, the flocks were vaccinated before any outbreaks of the disease began. However, there was one exception in the case of ranch 4. The sheep on this ranch were vaccinated after 35 ewes were dead. At least some losses were from black disease. This rancher had 104 ewes left. Fifty-two of them were vaccinated, and no deaths occurred within the group. But among the 52 controls, nine (17.3 per cent) died from black disease.

A summary of the results from the four ranches shows no death loss among the 173 immunized ewes. During the same time and under similar conditions, 16 (9.2 per cent) of the 171 unvaccinated controls died.

During the time the preceding experiments were in progress, an excellent opportunity was presented to test the value of the toxoid on 3,050 range sheep in which the infection was particularly active. Prior to the time when the losses were called to our attention at this ranch, an undetermined number of sheep had died. It is assumed that the majority of deaths were the result of black disease. However, from October 15, the owner kept an accurate record of all deaths. We feel fairly certain that all the losses discussed here represent only those sheep dead of this infection.

TABLE IX.—*Experimental vaccination of farm flocks.*

Flock	Vaccinated Sheep		Control Sheep		
	Total	Deaths	Total	Deaths	Loss (%)
1	24	0	23	2	8.7
2	33	0	32	1	3.1
3	64	0	64	4	6.2
4	52	0	52	9	17.3
Totals	173	0	171	16	9.2

Three groups of ewes were vaccinated on different dates so that the number of controls was not constant throughout the entire time during which the experiment was in progress. For this reason, table X has been subdivided to show the total number of sheep and the losses between the succeeding dates of vaccination. Since the number of controls was reduced by the number of animals vaccinated at each operation, the death loss has been computed upon a percentage per day basis rather than on a straight percentage basis.

The sheep included in the test were fine-wool range ewes of mixed ages. The vaccinated ones received the alum-precipitated toxoid prepared from the same strain used in the preceding tests. This strain proved to be serologically identical with the organism isolated from the sheep dying on this ranch. The toxoid was injected subcutaneously in one 5-cc dose. The data cover the period from October 15, 1937, to March 4, 1938, at which time the observations were discontinued, no losses having occurred after February 11.

There were 3,050 ewes on the ranch on October 15. Between this time and October 31, a period of 16 days, 13 sheep died (.027 per cent per day). On October 31,

111 ewes were vaccinated. No subsequent deaths occurred among the vaccinated group. However, deaths continued among the 2,926 non-vaccinated sheep at a slightly increased rate from October 31 to December 16. During this 46-day interval, there were 38 deaths, amounting to a 0.028 per cent loss per day. A second lot of 246 ewes was vaccinated on December 16. During the next 14 days, none of the 357 vaccinated sheep died. However, among the 2,642 controls, ten sheep died (0.027 per cent per day).

On December 30, 1,525 ewes were vaccinated. During the ensuing 24 days, December 30 to January 23, eight of the 1,882 vaccinated sheep died, giving a loss of 0.012 per cent per day. After January 23, there were no further losses in the vaccinated sheep, while deaths continued to occur in the controls until February 11. In the 43 days between December 30 and February 11, 15 of the 1,107 control sheep died (0.031 per cent per day). There were no losses in either the vaccinated sheep or the controls between February 12 and March 4, when observations were discontinued.

Table X shows the results of the experimental immunization of the range sheep. The total death loss from black disease in the control sheep was 76, while only eight sheep died in the vaccinated group. The percentage per day loss was constant in the control group throughout the black disease season. The only loss among any of the vaccinated sheep occurred during the first 24 days following the vaccination of 1,525 sheep on December 30, and the loss was confined to sheep vaccinated on that date. The losses occurred: one on the first day, two on the seventh, and one on each of the eighth, eleventh, 16th, 23rd and 24fh days.

It seems justifiable to assume that this loss would have been prevented if the sheep had been vaccinated before the black disease season, as some time is required after the use of any immunizing agent before full immunity is developed, this being particularly true of alum-precipitated products. Oxer[3] has shown that the antitoxic response in sheep immunized with his alum-precipitated anaculture was nil during the first week, with a high titre being reached between the second and fourth weeks, and the maximum response at three weeks.

TABLE X—*Experimental immunization of range sheep.*

Vaccination	Sheep Vaccinated	Observation Periods		Vaccinated Sheep	Loss		Control Sheep	Loss	
		1937–38	Days		Number	Per Cent per Day		Number	Per Cent per Day
		10–15—10–31	16				3,050	13	0.027
10–31	111	11– 1—12–16	46	111	0	0.0	2,926	38	0.028
12–16	246	12–17—12–30	14	357	0	0.0	2,642	10	0.027
12–30	1525	12–31— 2–11	43	1882	8*	0.01	1,107	15	0.031
		2–12— 3– 4	20	1874	0	0.0	1,092	0	0.0

*Loss occurred during the first 24 days following vaccination.

DISCUSSION

When it was recognized, in 1931, that death losses in farm flocks of sheep in the Bitterroot Valley were caused by the disease known in Australia as "black disease," which is a toxemia produced by *C. edematiens* following the invasion of the liver by immature flukes, the owners were advised to combat the primary invader by destroying the snails on their ranches. As little activity in fluke control developed, the possibility of immunization against the bacterial disease was investigated. At that time, the experimental evidence from Australia of the efficacy of immunizing agents was not convincing. Therefore, this laboratory started with work on products prepared according to Turner's methods, and eventually developed the alum-precipitated toxoid as an effective immunizing agent against black disease as it occurs in the field.

Before our work was completed, Oxer published the results of his work with an alum-precipitated anaculture. This type of immunizing agent has been found effective in Australia and is in general use there. There are several points in our work which may be emphasized as differing from the Australian work.

First, the product developed here is a toxoid, as compared with the anaculture used by Turner and by Oxer. As the death of the affected sheep is caused by toxemia, with only a small amount of tissue invasion by the organism, the stimulation of the production of antitoxin should be more important than the production of bactericidal antibodies. Experiments with guinea pigs indicated that this was the case, and the results of immunization of sheep in the field confirmed these findings.

An apparent advantage of the toxoid over the anaculture is that it may be used safely during an active outbreak of the disease. Turner warns against the use of the anaculture under such conditions, while our experience is that there is no aggravation of losses following immunization with the toxoid. The safety of the precipitated toxoid has been demonstrated in two outbreaks, one of which was particularly severe.

The second point in which our work differed from that of the Australian investigators is in the more successful controlled experiments with sheep, using artificial exposure to test immunity. The inoculation of sheep with cultures of *C. edematiens* does not produce a condition comparable to black disease as it occurs in the field. It was found, however, that intravenous injection of the toxin produced all the characteristic lesions of black disease except the liver lesions, which are caused by the invasion of flukes and bacteria. This method made it possible to test accurately in laboratory experiments the immunity of the sheep against an artificially-produced clinical condition comparable to that developing under natural conditions.

The importance of using a culture of high toxin-producing power for the production of an immunizing agent should be emphasized. This fact has been recognized by other workers. Turner,[1] using the unprecipitated anaculture, found the guinea pig mld must not be much larger than 0.005 cc for a 250-gm guinea pig (0.00,002 cc per gm). In our investigation, an anaculture having the initial guinea pig mld of 0.000,044 cc per gm stimulated only a very slight and inadequate immunity response. However, a satisfactory immunity was produced from a precipitated toxoid prepared from a culture which had a guinea pig mld of 0.00,005 cc per gram.

SUMMARY

The sheep disease known in Australia as infectious necrotic hepatitis, or black disease, has been recognized in western Montana, principally in farm flocks of sheep. The presence of the disease is manifested by sudden deaths from toxemia produced by the activity of *Clostridium edematiens* in the liver following invasion by immature flukes.

Investigations carried on simultaneously with similar investigations in Australia resulted in the development of an alum-precipitated toxoid prepared from a highly toxic strain of *C. edematiens*, the use of which prevents losses from this disease in the infected areas.

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Editorial

INFLUENZA

SPEAKING literally, influenza is well named. The word was coined by the Italian school of medicine during the 16th century. The physicians of Padua wrote better than they knew when they adopted this word to designate the systemic, febrile disease now known to be more a preliminary "influence" than a grave pathological entity, when not complicated by superimposed infections. The coming of the era of medical research, but a few years ago, did not furnish any reason for changing the name, for nowadays everyone understands that the "influence," then of unknown cause, is but the reaction to a somewhat benign ultramicroscopic agent that lays down a fertile medium for the growth of visible organisms, which are ever present and ready to invade the vital organs when the favorable moment arrives. In veterinary medicine, which is entitled to considerable credit for the modern conception of virus diseases, nothing is more revealing than the ultra-virus pathogens and the associated microbes which follow in their wake.

Influenza was one of the great plagues that overtook mankind during the 16th, 17th, 18th, 19th and 20th centuries. In 1889-1890, murderous epidemics swept from Asia Minor to as far west as the United States. And certainly, all will recall the deadly outbreak of 1918, which took more human lives at home than did the gunfire along the trenches of the Western Front. The lethal agent in these pandemics was not the virus of influenza alone. Visible bacteria striking the respiratory organs did most of the mischief.

In swine, influenza was first recognized as a definite disease in Iowa by Koen, of the United States Bureau of Animal Industry, during the World War. He named the disease "flu" and compared it to the human plague that was taking a tremendous toll throughout the country among the soldiers and civilians. Koen emphasized the benign character of the initial infection and the seriousness of the supervening complications where these were not curtailed by appropriate prophylactic measures. Today, this disease of swine is known to be a virus-bacillary complex. Shope, in this country, Verge, in France, and others have confirmed the original observation made by Koen among the swine herds of Iowa.

In dogs, it is not irrational to classify canine distemper as an influenza, for, here again, the somewhat harmless virus of Carré paves the way for the grave organic diseases caused by the bacterium of Ferry and M'Gowan (*Alkaligenes bronchisepticus*), which completely transfigures the clinical tableau. As a matter of fact, canine

distemper is comparable, pathogenically, to influenza. It, too, is a bacterial infection, developed under the depressing influence of an ultravirus. Its respiratory, intestinal and nervous manifestations are of bacterial, not of virus, origin.

In the equine genus, influenza is a similar disease. A virus, yet to be captured, is but the depressing agent which precedes the invasion of the visible organisms responsible for the organic complications which have placed this common malady among the existing plagues of horses. That there is an equine influenza virus is not questioned, notwithstanding that it remains to be isolated. Dieckerhoff, celebrated veterinarian of the 19th century, transmitted influenza to susceptible horses by inoculating them with whole blood of affected subjects; and others, before the filtration of viruses had become a fine art, transmitted the disease with defibrinated blood from which no demonstrable organisms could be isolated. This work of German veterinarians, coupled with the belief of Pasteur that there were living pathogens too small to be detected by the microscopes of their time, was the beginning of virus pathology.

There is no name, however, in English literature that is applicable to the contagious disease of horses and its multiple complications which we call influenza, shipping fever, catarrhal fever, pleuropneumonia, *et al.* It is a grave, disabling disease that strikes down horses agglomerated in markets, corrals, industrial establishments and on farms. Susceptibility is greatest in young horses, but the disease is no respecter of age when favorable conditions for its spread are created. The disease has been described under the names of equine influenza, distemper, strangles, typhoid fever, equine pasteurellosis, pinkeye, gourme (French), pferdestaupe (German), horse grippe, contagious pleuropneumonia of the horse, and other names too numerous to list here.

Dr. W. J. Embree, former chief veterinarian of the Western Weighing and Inspection Bureau,[1] regards irregular feeding in transit as a predisposing factor, claiming that the 28-hour law of the federal government is not reasonable for horses. Their digestive mechanism (relatively small stomach, long intestines, and large terminal reservoirs) lends itself only to a more or less continuous intake of food, any deviation from which leads to corporeal depression and broken-down resistance.

Outstanding authorities have pronounced the disease a primary infectious bronchitis and no one has yet been able to refute that simple hypothesis successfully. The part played by the alleged equine influenza virus of Dieckerhoff has not been positively confirmed, nor has the specificity of certain streptococci been proved. Basing his claims upon extensive observations made on military horses, Couillard[2] emphasizes the fact that neither the supposed ultravirus nor the microbes commonly isolated in the affected animals are entitled to be called specific agents. Quoting: "Until the specific agent of the disease is found, we can possess no specific treatment." In other words, mixed bacterins, symptomatic chemotherapy, and hygiene of the locale, ration and patient remain the treatments of choice.

The fact that the virus reaction of these infections is quite innocent and the subsequent bacterial invasion manifestly pronounced has removed much of the confusion that once surrounded their prophylaxis and treatment. But, what is still more interesting, from the veterinarian's point-of-view, is that much of the knowledge of virus diseases of plants and animals grew out of the work of animal pathologists, who have received scant credit for enriching the field of scientific investigation in that respect.

[1]Veterinary Military History of the United States, Vol. II, p. 970.

[2]Couillard, P.: Étude de l'evolution d'une epizootie de gourme et les moyens de lutte contre cette affection. (Thèse, Paris, 1937.) Abs., Rev. de Path. Comp., xxvii (1937), pp. 562-563.

TWO USEFUL CAREERS ENDED

On other pages of this issue of the JOURNAL are recorded the deaths of two famous veterinarians—Drs. Tait Butler and W. B. Niles. These two men had much in common. Both started their professional

careers in 1885. Both were identified with agricultural experiment station work for many years, Dr. Butler in Mississippi, Kansas and North Carolina, and Dr. Niles in Iowa and South Carolina. Both saw service in the U. S. Bureau of Animal Industry. Both were prominent in the A. V. M. A. for several decades. Both made priceless contributions to agriculture. Dr. Butler was called "the grand old man of southern agriculture." To Dr. Niles goes a large share of the credit for the development of anti-hog cholera serum, which has saved the swine industry millions of dollar every year for the past quarter of a century. Death has ended two useful careers.

MEMBERSHIP DIRECTORY

The Executive Board has authorized the publication of a new edition of the A. V. M. A. Membership Directory, and plans for getting out the new book are already under way. If you have not paid your dues for 1939, you should take care of this matter at once, for the following reason. Experience has shown that many members who change their addresses during the year do not report these changes promptly, but let the matter go until the next time they pay their A. V. M. A. dues. As evidence of this fact, we might mention that, during the past month, 187 members reported a change of address. A membership directory is only as valuable as it is accurate. You can help to make the new edition accurate by giving us your correct address. Will you coöperate?

A. V. M. A. EXHIBIT FUND

It is encouraging to be able to report this month quite a number of substantial contributions to the fund being raised to finance a veterinary exhibit at the coming New York World's Fair. Dr. J. R. Mohler, who is chairman of the special committee having this project in charge, recently announced that it had been necessary to contract for some additional space in order to take care of the exhibit adequately. The additional subscriptions to be reported this month are as follows:

District of Columbia Veterinary Medical Association	$ 25.00
Dr. Charles J. Frey, Silver Springs, Md.	5.00
Iowa Veterinary Medical Association	250.00
Michigan State Veterinary Medical Association	50.00
South Dakota Veterinary Medical Association	25.00
Southwestern Minnesota Veterinary Medical Association	25.00
Vermont Veterinary Medical Association	10.00
Western New York Veterinary Medical Association	50.00

The subscriptions previously reported* amounted to $947.50. The eight contributions listed above bring the total to $1,387.50.

INVITATIONS FOR 1941

The American Veterinary Medical Association will meet in Memphis, Tenn., this year and it already has been decided to hold the 1940 convention in Washington, D. C., both of these selections having been made at the 1938 meeting in New York City.

In keeping with plans to continue selecting each meeting place two years in advance, instead of one year ahead as has been customary in the past, it will be in order for us to decide at the Memphis meeting where we will hold our 1941 convention.

The plans now being formulated provide for dividing the United States and Canada into a number of zones or districts, with a view to rotating conventions throughout these zones or districts, according to a schedule to be decided upon later. Tentatively, the following states have been selected to make up a central zone or district: North Dakota, South Dakota, Nebraska, Kansas, Minnesota, Iowa, Missouri, Wisconsin, Illinois, Michigan, Indiana, Kentucky, Ohio and West Virginia.

In all probability, there will be three other convention zones or districts, one to comprise the western states, one the south-

(*Continued on page* 136)

*Journal of the A. V. M. A., January, 1939, p. 5.

APPLICATIONS FOR MEMBERSHIP

This month we are giving first listing to 46 applications, the largest number to be listed in any month since August, 1938. Comparing the listings this month with the corresponding month of recent years, we find that in February, 1938, there were 41 applications listed and 43 in the same month of 1937. It is necessary to go back to 1929 to find the previous record number for February—45. Therefore, February of 1939 establishes a new record for the month.

FIRST LISTING

(See January, 1939, JOURNAL)

ALLEN, RAYMOND RIPLEY
365 Pleasant St., Leominster, Mass.
D. V. M., Cornell University, 1932. Vouchers: L. A. Paquin and W. H. Dodge.

ARLEIN, MYRON SAUL
Box 432, Harrisonburg, Va.
D. V. M., Iowa State College, 1938. Vouchers: Don A. Yandell and H. D. Bergman.

BARTLEY, THEODORE F.
Vesta, Minn.
D. V. M., Iowa State College, 1933. Vouchers: Carl Hansen and L. E. Stanton.

BASTIAS NEIRA, OCTAVIO
Casilla 537, Santiago, Chile.
M. V., Universidad de Chile, 1932. Vouchers: Julio San Miguel and L. A. Merillat.

BENNETT, MARCUS ERVING
Neillsville, Wis.
D. V. M., Chicago Veterinary College, 1917. Vouchers: James S. Healy and William R. Winner.

CARLISLE, BILLY ELZA
Camilla, Ga.
D. V. M., Alabama Polytechnic Institute, 1917. Vouchers: J. E. Severin and Chas. C. Rife.

CLARK, AUGUSTUS B.
336 Post Office Bldg., Oklahoma City, Okla.
D. V. M., Alabama Polytechnic Institute, 1934. Vouchers: L. J. Allen and Wilbur McPherson.

CLAY, CHARLES C.
Box 3035, Cristobal, Canal Zone.
D. V. M., McKillip Veterinary College, 1917. Vouchers: Lt. Col. Francois H. K. Reynolds, L. A. Merillat and T. L. Casserly.

COLE, ELVIN G.
31, Public Safety Bldg., Salt Lake City, Utah.
D. V. M., Colorado State College, 1936. Vouchers: W. H. Hendricks and Jean C. Flint.

CRISPELL, THOS. P.
Parsons, Kan.
D. V. M., Chicago Veterinary College, 1920. Vouchers: Chas. W. Bower and R. F. Coffey.

CROW, LOUIS C.
336 Post Office Bldg., Oklahoma City, Okla.
D. V. M., Kansas City Veterinary College, 1915. Vouchers: L. J. Allen and Wilbur McPherson.

COATES, MAX SIBLEY
North Lewisburg, Ohio.
D. V. M., Ohio State University, 1933. Vouchers: W. F. Guard and C. R. Donham.

COVER, MORRIS S.
Durham, N. H.
V. M. D., University of Pennsylvania, 1938. Vouchers: C. L. Martin and C. A. Bottorff.

CUMMINGS, JAKE C.
336 Post Office Bldg., Oklahoma City, Okla.
D. V. M., Kansas City Veterinary College, 1917. Vouchers: L. J. Allen and Wilbur McPherson.

DICTOR, JOSEPH
1415 Main St., Lynchburg, Va.
V. M. D., University of Pennsylvania, 1937. Vouchers: F. E. Lentz and G. A. Dick.

DUDLEY, KENT RUGGLES
Iola, Kan.
D. V. M., Kansas State College, 1923. Vouchers: Chas. W. Bower and R. F. Coffey.

FINK, CARL
336 Post Office Bldg., Oklahoma City, Okla.
D. V. M., Texas A. and M. College, 1938. Vouchers: L. J. Allen and Wilbur McPherson.

FOODER, ELWOOD GODFREY
110 Haddon Ave., Haddonfield, N. J.
V. M. D., University of Pennsylvania, 1935. Vouchers: R. A. Hendershott and J. G. Hardenbergh.

FRANCISCO, DONALD JAMES
13125 Hamilton Ave., Detroit, Mich.
D. V. M., Michigan State College, 1938. Vouchers: S. R. Elko and N. C. Laughlin.

FREDRICKSON, LUTHER ERICK
814 15th St., Highland, Ill.
D. V. M., Michigan State College, 1937. Vouchers: C. F. Clark and E. T. Hallman.

HEDGE, RUSSELL H.
Hedrick, Iowa.
D. V. M., Iowa State College, 1920. Vouchers: P. V. Neuzil and L. M. Hadley.

HEMPHILL, JOHN GRAHAM
317½ White St., Norman, Okla.
D. V. M., Kansas State College, 1937. Vouchers: S. E. Douglas and L. J. Allen.

HOUCHIN, E. H.
155 S. Ventura Ave., Ventura, Calif.
B. S., D. V. M., State College of Washington, 1924. Vouchers: W. L. Curtis and John L. Tyler.

IRWIN, IRVIN P.
336 Post Office Bldg., Oklahoma City, Okla.
D. V. M., Kansas State College, 1937. Vouchers: L. J. Allen and S. E. Douglas.

KAPLAN, SIDNEY L.
543 East 169th St., New York, N. Y.
D. V. M., Ohio State University, 1938. Vouchers: C. R. Donham and W. R. Krill.

LAWRENCE, WILLIAM ROSS
Forked Deer Hotel, Dyersburg, Tenn.
D. V. M., Saint Joseph Veterinary College, 1923. Vouchers: John H. Gillmann and E. B. Mount.

LUITHLY, WILLIAM E.
Richland, Iowa.
D. V. M., Iowa State College, 1912. Vouchers: N. L. Nelson and P. V. Neuzil.

MC CORMICK, LT. EDMUND BURKE
CCC Hdqrs., Sacramento, Calif.
D. V. M., Kansas State College, 1937. Vouchers: W. L. Curtis and Geo. M. Wilson.

MAICHEL, GEORGE B.
Overbrook, Kan.
D. V. M., Kansas State College, 1938. Vouchers: Edwin J. Frick and R. F. Coffey.

MERKLEY, MYRT E.
Saint Peter, Minn.
V. S., B. V. Sc., Ontario Veterinary College, 1938. Vouchers: E. H. Gloss and C. W. Olson.

MINSKY, LAWRENCE
2763 W. Valley Blvd., El Monte, Calif.
D. V. M., Iowa State College, 1937. Vouchers: W. L. Curtis and Geo. M. Wilson.

MOHR, CHARLES ADAM
1624 North Elwood, Tulsa, Okla.
D. V. S., Kansas City Veterinary College, 1911. Vouchers: L. J. Allen and S. E. Douglas.

PAUL, GEORGE J.
Saint Cloud, Minn.
D. V. M., McKillip Veterinary College, 1918. Vouchers: E. H. Gloss and C. P. Fitch.

PINT, LAWRENCE H.
Austin, Minn.
D. V. M., Iowa State College, 1936. Vouchers: E. H. Gloss and L. E. Stanton.

PLUMMER, W. B.
Millersburg, Ky.
D. V. M., Indiana Veterinary College, 1938. Vouchers: Floyd E. Hull and T. P. Polk.

RODRIGUEZ TORO, RAMON
Casilla 1641, Santiago, Chile.
M. V., Universidad de Chile, 1933. Vouchers: Julio San Miguel and L. A. Merillat.

SANZ GARCIA, BALBINO
Casilla 537, Santiago, Chile.
Escuela Superior de Veterinaria, Madrid, Spain, 1907. Vouchers. Julio San Miguel and H. Preston Hoskins.

SAULMON, ERNEST, E.
Box 190, Bowling Green, Ky.
D. V. M., Ohio State University, 1935. Vouchers: D. E. Westmorland and T. P. Polk.

SCHAAF, KERMIT
Kimber Poultry Breeding Farm, Niles, Calif.
D. V. M., Michigan State College, 1932. Vouchers: W. L. Curtis and Nelson E. Clemens.

SMITH, ROY C.
421 S. Van Buren, Enid, Okla.
D. V. M., Kansas City Veterinary College, 1914. Vouchers: L. J. Allen and Wilbur McPherson.

STABB, JOHN J.
16 Prospect St., Montpelier, Vt.
D. V. M., McKillip Veterinary College, 1914. Vouchers: G. N. Welch and Albert F. Ranney.

STAMEN, HARRY C.
Woodside Park, New Rochelle, N. Y.
D. V. M., Ohio State University, 1922. Vouchers: Orville E. McKim and C. E. DeCamp.

TIMM, F. W.
3664 E. Layton Ave., Cudahy, Wis.
B. V. Sc., Ontario Veterinary College, 1936. Vouchers: L. A. Merillat and Cecil Houston.

VOETBERG, HENRY G.
336 Federal Bldg., Oklahoma City, Okla.
D. V. M., Iowa State College, 1931. Vouchers: L. J. Allen and Wilbur McPherson.

VAN WAGENEN, VICTOR F.
Wallkill, N. Y.
D. V. M., Cornell University, 1937. Vouchers: H. J. Milks and R. J. Garbutt.

WELSH, DONALD R.,
104 Wood St., Greenfield, Ind.
D. V. M., Indiana Veterinary College, 1915. Vouchers: J. L. Axby and L. A. Merillat.

Applications Pending

SECOND LISTING

(See January, 1939, JOURNAL)

Allison, Lt. Aaron Francis, Federal Bldg., Memphis, Tenn.
Bixby, John S., 531 S. Holmes St., Memphis, Tenn.
Cunningham, Charles Henry, University of Maryland, College Park, Md.
Denney, Paul C., 27 S. Barksdale, Memphis, Tenn.
Digranes, Joseph Haakon, 336 Federal Bldg., Oklahoma City, Okla.
Edmondson, Harry K., 1801 Homan Ave., Fort Worth, Tex.
Hatch, Ray D., Iowa State College, Ames, Iowa.
Johnson, Klemens Frank, 511 University Ave., Laramie, Wyo.
Jones, Carrel L., 1435 Murphy's Lane, Salt Lake City, Utah.
Kallenberg, Edwin Frank, Union City, Tenn.
Kime, William, 613 Dominion Public Bldg., Winnipeg, Man., Can.
Lucas, J. W., Abingdon, Ill.
Mackenzie, William Parker, Franklin Roads, Maine.
Nichols, Wilbert C., Box 447, Payette, Idaho.
Pundt, Werner, 1709 East Main St., Medford, Oregon.
Ruth, Vincent W., Box 1671, Charleston, W. Va.
Soldner, Paul Albert, State and North Sts., Dover, Del.
Wilson, John Thomas, Pawnee, Okla.
Yabsley, Francis Charles, Cissna Park, Ill.

The amount which should accompany an application filed this month is $9.58, which covers membership fee and dues to January 1, 1940, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Atlanta Veterinary Medical Society. Atlanta Athletic Club, Atlanta, Ga. February 1, 1939. Dr. Chas. C. Rife, Secretary, 420 Edgewood Ave., Atlanta, Ga.

Connecticut Veterinary Medical Association. Hotel Bond, Hartford, Conn. February 1, 1939. Dr. Geo. E. Corwin, Secretary, State Office Bldg., Hartford, Conn.

New York City, Veterinary Medical Association of. Town Hall, New York, N. Y. February 1, 1939. Dr. C. R. Schroeder, Secretary, New York Zoölogical Park, Bronx, N. Y.

Missouri Veterinary Medical Association and University of Missouri Veterinary Short Course. University of Missouri, Columbia, Mo. February 1-3, 1939. Dr. C. L. Campbell, Secretary, 7224 Tulane St., Saint Louis, Mo.

Dallas-Fort Worth Veterinary Medical Society. Fort Worth, Texas. February 2, 1939. Dr. H. V. Cardona, Secretary, 2736 Purington Ave., Fort Worth, Texas.

Houston Veterinary Association. Houston, Texas. February 2, 1939. Dr. E. G. Pigman, Secretary, 4206 Polk Ave., Houston, Texas.

Small Animal Hospital Association, Los Angeles, California. February 7, 1939. Dr. R. W. Gerry, Secretary, 8474 Melrose Ave., Los Angeles, Calif.

Hudson Valley Veterinary Medical Society. Poughkeepsie, N. Y. February 8, 1939. Dr. J. G. Wills, Secretary, Delmar, N. Y.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. February 8, 1939. Dr. J. P. Torrey, Secretary, 610 Veronica Ave., East Saint Louis, Ill.

Southeastern Michigan Veterinary Medical Association. Medical Arts Building, 3919 John R St., Detroit, Mich. February 8, 1939. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. February 14, 1939. Dr. O. Norling-Christensen, Secretary, Box 12, Wilmette, Ill.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. February 15, 1939. Dr. Charles Eastman, Secretary, 725 S. Vancouver Ave., Los Angeles, Calif.

Kansas Veterinary Medical Association. Veterinary Division, Kansas State College, Manhattan, Kan. February 15-16, 1939. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kan.

Illinois State Veterinary Medical Association. Leland Hotel, Springfield, Ill. February 16-17, 1939. Dr. C. C. Hastings, Secretary, Williamsville, Ill.

Manitoba, Veterinary Association of. Marlborough Hotel, Winnipeg, Man., Can. February 17, 1939. Dr. Wm. Hilton, 411 McIntyre Block, Winnipeg, Man., Can.

Kansas City Veterinary Medical Association. Kansas City, Mo. February 20, 1939. Dr. S. J. Schilling, Secretary, Box 167, Kansas City, Mo.

San Diego County Veterinary Medical Association. Zoölogical Research Bldg., Balboa Park, San Diego, Calif. February 20, 1939. Dr. Glenn A. Tucker, Secretary, Vista, Calif.

Colorado State College Veterinary Short Course. Fort Collins, Colo. February 21-23, 1939. Dr. Jas. Farquharson, Colorado State College, Fort Collins, Colo.

Alabama Veterinary Medical Association and Short Course for Graduate Veterinarians. College of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala. February 21-24, 1939. Dr. I. S. McAdory, Secretary, Alabama Polytechnic Institute, Auburn, Ala.

Keystone Veterinary Medical Association. School of Veterinary Medicine, 39th St. and Woodland Ave., Philadelphia, Pa. February 22, 1939. Dr. C. S. Rockwell, Secretary, 4927 Osage Ave., Philadelphia, Pa.

Massachusetts Veterinary Association. Hotel Westminster, Copley Square, Boston, Mass. February 22, 1939. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Louisiana Veterinary Medical Association and Louisiana State University Veterinary Short Course. Dalrymple Memorial Bldg., Louisiana State University, Baton Rouge, La. February 22-23, 1939. Dr. C. M. Heflin, Secretary, Box 1933, Baton Rouge, La.

Virginia State Veterinary Medical Association. Virginia Polytechnic Institute, Blacksburg, Va. March 7, 1939. Dr. A. J. Sipos, Secretary, 1102 State Office Bldg., Richmond, Va.

Virginia Polytechnic Institute, Conference for Graduate Veterinarians at. Blacksburg, Va. March 7-9, 1939. Dr. I. D. Wilson, Department of Zoölogy and Animal Pathology, Virginia Polytechnic Institute, Blacksburg, Va.

American Animal Hospital Association. Miami Biltmore Hotel, Miami, Fla. March 23-25, 1939. Dr. D. A. Eastman, Secretary, 901 - 19th St., Moline, Ill.

State Board Examination

Virginia Board of Veterinary Examiners. March 8, 1939. Virginia Polytechnic Institute, Blacksburg, Va. Further information may be obtained from Dr. H. H. Adair, Secretary, 107 Water St., Bristol, Va.

Ravenous Wolves Kill Reindeer in Finland

Wolves, starved and roaming in packs in search of food, have killed nearly 1,000 reindeer in northern Finland. According to a report issued early in January, armed expeditions, organized by the government, are helping to exterminate the wolves.

AMERICAN ANIMAL HOSPITAL ASSOCIATION

The annual meeting of the American Animal Hospital Association will be held in Miami, Fla., March 23-25, 1939. Secretary Eastman advises that a program of outstanding interest has been arranged and this, together with an opportunity to visit tropical Florida, at a time of the year when all of the winter activities are still in full swing, should prove to be an attraction difficult to resist.

Transportation rates to Florida at this time of the year are very reasonable, and hotel and other accommodations will be available at rates no greater than those charged at meetings in other parts of the country.

The Republic of Cuba has extended an invitation to those in attendance at the meeting to make a post-convention trip to Havana. The latest available reports are to the effect that tentative plans have been made to accept the invitation.

If you want to receive a program of the meeting, send your request to Dr. D. A. Eastman, Secretary, 901 - 19th St., Moline, Ill.

Blood Potassium in Colics

As the reactions of animals and man to the same disease differ, chemotherapy based exclusively upon animal experimentation may be faulty when applied to man, and vice versa. An example is the increase of blood potassium in animals affected with intestinal obstruction compared with a decrease in human patients so affected. This mathematically opposite state was discovered by workers at the Mayo Clinic, where therapeutic finesse accounts for the beaten path to Rochester. While predicating this finding to the baffling digestive troubles of animals (colics) is not insisted upon, it does open an undeveloped field of investigation in the domestic herbivores, where little or nothing is known of the effect of intestinal obstruction on the elements of the blood.

ACANTHOSIS NIGRICANS: REPORTS OF FOUR CASES*

By HERMAN STARK, *Brooklyn, N. Y.*

While the scarcity of authoritative publications on acanthosis nigricans would seem to indicate that this condition is a comparatively rare one, my own experiences, during the past two years or more, would suggest that the disease is more common than is generally believed.

In the hope that my observations and the line of treatment which was adopted very successfully in all cases coming to the Brooklyn Veterinary Hospital for treatment during this period will be of interest and value to my professional colleagues, I am pleased to submit the following comments and report on a series of four cases, although six more cases are recorded at our hospital.

Acanthosis nigricans, also known as keratosis nigricans or papillary and pigmentary dystrophia, is characterized by a symmetrical, bilateral hypertrophy of the papillary bodies and moderate thickening of the epidermis, especially in the furrows of the skin. There is an increase of pigment in both layers of the epidermis and, unlike a similar disease in man, in the papillary bodies around the blood-vessels and glands.

It affects chiefly the axillae, the folds of the stifle, the under-surface of the neck, the extensor side of the toes, the region of the anus and scrotum, the abdomen and the angles of the mouth, eye-lids and lips.

Its effects are manifested first by a swelling of the skin and shedding of the hair. The skin, which becomes unusually soft, with furrowing more pronounced than usual, takes on the appearance of seal-skin leather. This is followed by the development of a grayish-blue or blackish pigmentation. Numerous papillary swellings appear and the skin is criss-crossed with ridges or folds. It becomes harder and rougher than normal.

Although itching is usually present, there is little exfoliation and only rarely slight desquamation. The disease may spread to the claws or to adjacent mucous membranes, where soft, red granulations appear. These do not bleed.

A study of my case records emphasizes the need of an accurate differential diagnosis. The history of several of these cases, which came to me with lengthy records of unsuccessful treatment, indicates only too clearly that the disease is not always recognized. Consequently, in such cases the most effective treatment was not employed and results were disappointing to the pet-owners and presumably to the animals themselves.

The differential diagnosis should exclude chronic eczema, pruritus and follicular mange. In this latter condition, the characteristic symmetrical pigmentation of acanthosis nigricans is absent, pigment is contained only in the superficial layers of the skin and comes away with it, and the Demodex mite may be demonstrated in the skin scrapings.

It is worthy of note that no known cause for the disease has been discovered, but I might mention here that the successful results which I have obtained with colloidal sulfur, as reported below, might point to some local sulfur deficiency as being responsible in some way. I am not prepared at this time to go any farther than just to suggest such a possibility.

Contrary to the opinion generally expressed in such publications as are available

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up to this time, my experience has shown that acanthosis nigricans is not usually confined to young dogs but is found in dogs of all ages.

It is naturally difficult to generalize on the basis of only a few cases but, as will be observed from their histories, no age partiality seems to be very evident.

CASE 1

Male, wire-hair terrier, eight years old. The owner brought him to the Brooklyn Veterinary Hospital on March 4, 1936, stating that, some four or five months earlier, he had noticed changes in the appearance of the skin on certain parts of the dog's body which had become progressively worse until, as he stated, "the skin under his neck and on his belly looks all blackish and discolored."

The dog, which had been treated, since the condition was first noticed, by four different veterinarians without success, was left at the hospital but with little hope on the part of his owner.

The dog had been fed a diet which was substantially correct and was in fair general condition although he had lost a little weight. There was little apparent irritation and itching.

Examination disclosed a seal-leather-like condition of most of the areas in which acanthosis nigricans is usually found. There was the characteristic symmetrical bilateral pigmentation, which seemed to rule out follicular mange. This condition was definitely excluded when examination of skin scrapings showed the absence of the Demodex mite. A definite diagnosis of acanthosis nigricans was made and treatment instituted immediately with colloidal sulfur in lotion form (collo-sul).

The lotion was applied liberally on cotton swabs three times a day. The affected skin seemed to act just like a sponge and sufficient lotion was applied to saturate the parts as nearly as possible. No covering is necessary, as colloidal sulfur is not poisonous. Even if the dog should be able to imbibe some of the lotion by licking the parts after treatment, it will act only as a mild aperient. This colloidal sulfur lotion is not irritating.

No other treatment, beyond giving the animal a normal adequate diet, was used. The condition began to improve immedi-

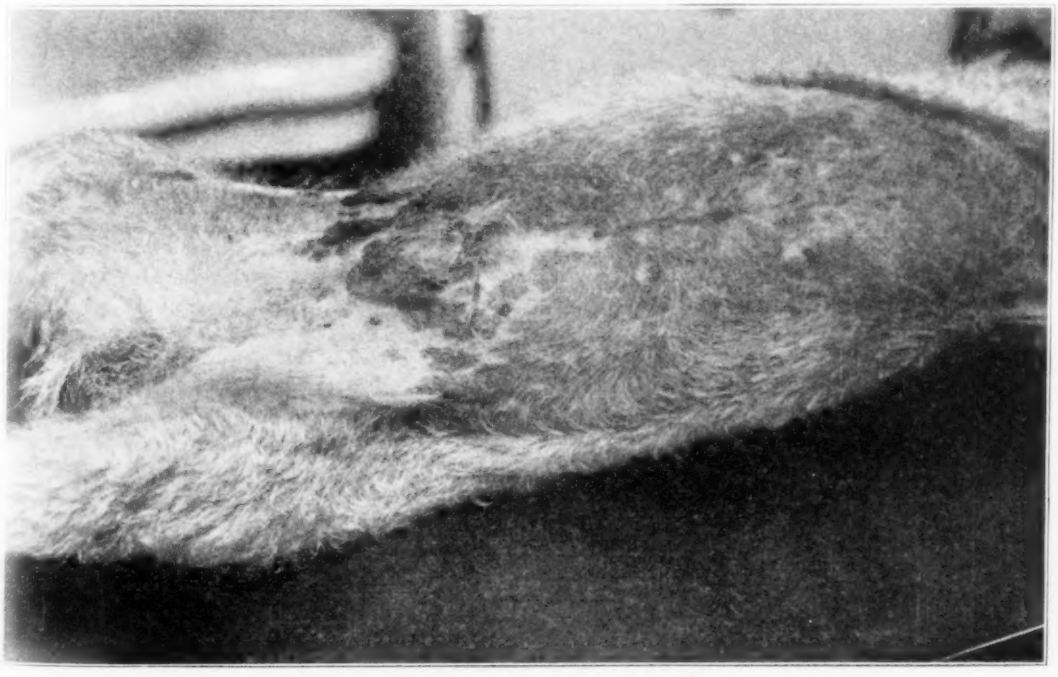

FIG. 1. Case 1, on September 24, 1938, 2½ years after treatment.

ately, the hair grew again and the dog was returned to his owner, completely cured, on March 24.

CASE 2

Male, Scottish terrier, three and one-half years old, left at the hospital on July 11, 1938, by his owner, who gave a history similar in almost every way to that of case 1. This dog had been treated for over seven months by four other veterinarians but without tangible results.

The dog was in fair general condition, had apparently been fed a substantially correct diet and had lost only a little weight. The hair had been shed from the underside of the neck, the axillae, the abdomen and from the area around the anus and scrotum. These parts looked blackish and were hard and rough and covered with numerous little condylomatous swellings. The appearance of the skin and the location of the parts affected suggested acanthosis nigricans, while absence of the Demodex mite from the skin scrapings excluded follicular mange.

Treatment with colloidal sulfur lotion was immediately instituted, exactly as in case 1, and the response was equally prompt. The dog was discharged, completely cured, on July 30, after only 19 days of treatment.

CASE 3

Male, Airedale terrier, age twelve years. This case is particularly interesting in view of the age of the dog.

A definite diagnosis of acanthosis nigricans was made, follicular mange being excluded by examination of skin scrapings. The dog had been affected for over two years and two other veterinarians had not succeeded in curing the condition.

Treatment with colloidal sulfur lotion was started on February 6, 1937, and the dog was discharged, completely cured, on February 22.

CASE 4

Male, Scottish terrier, four years old. This dog presented the characteristic symptoms of acanthosis nigricans, a definite diagnosis being made after elimination of demodecosis.

The condition had persisted for over nine months and had been treated unsuccessfully by two other veterinarians.

Treatment with colloidal sulfur lotion was instituted immediately and the condition cleared up so rapidly that discharge was possible within the very short time of eight days. The owner was allowed to continue treatment at home. However, regular examinations were made over a period of two weeks. The lesions have entirely disappeared, with no recurrence of the disease to date.

CONCLUSIONS

These case reports indicate the satisfaction which can be obtained in the treatment of this condition when a definite differential diagnosis is made and I believe the report will be interesting in view of the simplicity of the treatment used.

Acanthosis nigricans should be differentiated from follicular mange and chronic eczema.

It is of paramount importance for the examining veterinarian to recognize the disease.

A colloidal sulfur lotion seems to have the advantage of reliable efficiency, which was certainly not characteristic of methods heretofore employed.

It might be worthy of mention at this time that I have had excellent results in other skin conditions, in which sulfur is indicated, with the particular lotion referred to.

SPECTACULAR POISONING WITH A MORAL*

By JAMES W. BENNER
State College, New Mex.

On October 4, 1938, a call for help came to the Extension Service from one of the large cattle companies in the state. The previous afternoon, this company had vaccinated and branded calves in one of their units located close to the Mexican border. In three to four hours, in a group of 17 head, three to four weeks of age, some deaths had occurred and the rest were very

*Received for publication, November 5, 1938.

sick. Some of the larger calves, four and five months of age, also were sick. By the following morning, eight to twelve hours after vaccination and branding, the 17 head of smaller calves and a few of the larger ones were dead.

The camp that was working the cattle consisted of 20 men, 18 riders, one cook, and one wrangler. All vaccinating and branding was stopped when the calves began to die. A diagnosis of the trouble and the cause was very necessary before operations could be continued.

The ranch superintendent had consulted the representative of the serum company who had sold the biologics used in the vaccination. The representative from the serum company and the writer drove to the ranch to investigate the losses. We arrived at ranch headquarters too late Tuesday evening to examine the sick and dead animals. We spent the evening talking about the history of the cases and the possibilities regarding the trouble and the cause. We learned that the routine procedure with the calves was, first, hypodermic injections subcutaneously of 1 cc of blackleg bacterin and 3 cc of pulmonary mixed bacterin, given from separate syringes, at separate points on the animal. The second step was the branding, which consisted of three brands on the left side, one on the hip, one on the body and one on the jaw. Third, the newly made brands were smeared with a pine-tar preparation and linseed oil, mixed in proportions of about 40 per cent of the pine-tar preparation to about 60 per cent of the linseed oil. This dressing was used to promote healing and protect from screw-worms.

The symptoms which had been observed were marked distress, restlessness, salivation and drooling, diarrhea, prostration and death.

We did not think it probable that any bacterial contamination in the biologics used in the small dosages (1 cc and 3 cc) would cause such sudden and heavy losses. We thought the cause must be chemical and not bacterial.

Shock from the branding was pretty well eliminated because it had not killed calves before. The same was true of the vaccination. In fact, some of the same lot of bacterin had been used without harmful effects. Therefore, two things seemed fairly clear. One was that the cause was chemical in nature and that it was very likely in the preparation smeared on the newly made brands. When the ranch superintendent asked what chemicals might have caused the losses, several were enumerated, arsenic included. At the mention of arsenic, the ranch superintendent remembered that the mixture of pine tar and linseed oil for the Monday afternoon branding was especially dark, but, upon smelling it, the odor seemed the same as usual.

Upon questioning his men about the source of the ingredients of the mixture, he had learned that the so-presumed linseed oil for the mixture had been taken out of an old five-gallon can, which had been around the camp for about 14 years. The label on the can was very nearly obliterated and all positive knowledge of the contents forgotten. The man who had taken out some of the fluid thought it looked like linseed oil and had used it in about a 60 per cent proportion with the pine-tar preparation. In thinking it over, the superintendent thought that the can might have contained some arsenical dip.

The observation of this incident by the ranch superintendent and his perfect honesty in telling us of it gave us a clue that made it easy to gather further evidence which would clinch the evidence that would "convict the calf-killer," that is, make a diagnosis of the trouble and a determination of the cause possible.

The ranch superintendent was asked if he would risk the loss of one, two, or possibly three more calves for a test in order to determine definitely what part of the Monday afternoon operations had caused the losses. He replied that he was perfectly willing, because they would not continue the vaccinating and branding until they knew definitely the nature of the trouble and how to avoid it.

Therefore, we suggested that the next morning (Wednesday) we go to the camp

early and take three calves, the same size as the 17 that had died, and run the following test:

Calf 1—Give the vaccination only.

Calf 2—Give the vaccination plus the three brands.

Calf 3—Give the vaccination plus the branding plus the smearing with the same tar mixture that had been used.

This was agreed upon and, the next morning, I drove with the ranch superintendent and the serum company representative to the cow-camp, about six miles south of headquarters. We reached the camp just as the cowboys were picking out their mounts for the first ride of the day. They had rounded up the herd of about 135 head and were making a temporary enclosure with a single rope held by men at close intervals, while other men picked out the horses to be ridden.

We were informed that it would take probably an hour or two to bring the three calves for the test and their mothers off the range. We spent the time looking over a herd of cows and some of the older calves, five or six months of age, which had been vaccinated, branded and smeared. On some, the branding and smearing had been done on the jaw only. These calves had been branded on the hip and body at some previous time.

The animals showed some local swelling on the jaw, extending down to the throat. But there were no noticeable symptoms of a general systemic disturbance. One of the larger calves, which had received the vaccination plus the brand and smearing on the jaw and body, showed a slight swelling on the jaw and a diarrhea, and was slightly droopy but not dangerously sick.

Another of the larger calves, which had received the vaccination and three brands smeared with the tar mixture, showed a little swelling on the jaw and serious systemic disturbance. He was not nursing, but showed congestion of the mucous membranes and an unsteady gait. Three or four like this calf, which had received the same treatment, had died the night before (Tuesday). All of these showed a purplish discoloration of the mucous membranes of the mouth, nose and eyes. A severe diarrhea was manifested by the soiled tails and, in some cases, by blood-stains, which indicated a bloody diarrhea before death.

A peculiar condition which had been observed by the ranch superintendent and observed again on this morning was that all dead calves lay on the branded side. The only theory that came to mind was that the pain had probably induced them to take their final position on this side. Still another medium-sized calf, weighing about 250 pounds, was very sick. This calf was lying down and breathing fast, showed diarrhea and congested mucous membranes and, when lifted to his feet, wabbled toward a waterhole but did not drink. His temperature was 101° F. In a few hours, before noon, this calf was dead. On post-mortem examination, the principal change found was an inflammation of the digestive tract, or gastroenteritis.

When the three cows and their calves had been run into a small corral and the branding-irons had been heated, we were ready to start our test. The men who had done the work on Monday were to do it again, using the same technic. As previously agreed upon, calf 1 was vaccinated; calf 2 was vaccinated and branded; and calf 3 was vaccinated, branded and smeared with the same mixtures that had been used Monday afternoon.

It was 8:45 a. m. when we finished with calf 3. Observations were made and recorded as follows: At 9:00 a. m., the respirations of calf 3 increased; the animal was panting like a dog. Calves 1 and 2 remained normal. Five minutes later, calf 3 lay down and saliva started to run from the mouth. At 9:10 a. m., the animal arouse but, ten minutes afterward, with the tail lifted and some evidence of straining, it lay down for the second time. At 9:30, the head was turned to one side, resting on the ground. At 9:40, we forced the animal to get up. The gait was slow and wabbly and diarrhea was observed. Five minutes later, calf 3 lay down for the third time. The ears were down and the animal was very droopy, breathing fast, with the head resting on the ground.

At 9:50, calf 1 nursed normally; no change was noted. Calf 2 also seemed normal, and there was no change. Calf 3, at 10:00, was breathing irregularly and had lain down for the fourth time, remaining on the ground while its temperature, which registered 100.6° F., was being taken. At 10:10, calf 2 nursed normally. Both calves 1 and 2 were obviously normal. Five minutes later, we again forced calf 3 to get up. The animal was very wabbly on its feet, the muscles of the shoulders twitched, and the gait was slow and unsteady.

At 10:20, calf 3 lay down again, this time flat on the ground, with the face downward, breathing slowly. At the end of another ten minutes, the animal was barely able to get up. It lay down immediately after the attempt, prone and showing the slow, hard, abdominal type of breathing. The temperature was 98.4° F.

At 10:45, two hours after treatment, the animal lay on the ground, unconscious. The mouth was open, the mucous membranes were congested, and very thin feces were being passed. This condition remained until death, which occurred at noon, just three hours and 15 minutes after the vaccination, branding and smearing. The principal postmortem change was a gastroenteritis. Throughout this period, calves 1 and 2 remained normal.

With difficulty, we learned from the label of the old five-gallon can, from which the supposed linseed oil had been taken, that the original contents was arsenic dip. Evidently the can still contained some of the dip in a very concentrated form. The diagnosis given was arsenic poisoning and, since, it has been confirmed by chemical analysis. The cause was the mistaken substitution of arsenic dip for linseed oil in a tar preparation used on freshly made brands.

The moral, which is the most impressive I have ever seen, is that ranchers, farmers, doctors and all others should keep the correct labels on cans, bottles and other containers, or destroy them. Taking a chance on an unknown substance may cause disastrous results, as it did in this case.

WILD-CHERRY POISONING IN PIGS*

By E. H. ENGE, *Comfrey, Minn.*

On July 5, 1938, I was called to see some pigs which I had immunized against hog cholera on June 29. Naturally, upon receiving a call of this nature, a practitioner in the Corn Belt would become extremely alarmed, especially when the pigs appeared to be healthy at the time of immunization.

History: I had immunized 95 pigs on the above-mentioned date. The owner had been very careless about following feeding directions after immunization. Three pigs were taken sick approximately twelve hours before I was called on July 5. The 95 were being pastured in a large orchard.

Symptoms: Temperature was subnormal and two of the pigs were sluggish, while one had convulsions, staggered, had dilated pupils and breathed with difficulty. The others were in the same condition at the time of immunization. We autopsied the one which was about ready to succumb to the poisoning.

Lesions: Blood was of a cherry-red color. Gastroenteritis was present along with lesions due to cholera immunization.

Diagnosis: I immediately suspected poison, but to convince my client that the trouble was not a so-called serum break was a different matter. We started searching about the orchard for poisonous plants. Finally we ran across small trees where the pigs had been resting. We noticed where they had been eating leaves and also some roots. I advised him to take the pigs out of the orchard, which he did, and the trouble was checked immediately. The two pigs recovered after symptomatic treatment, as they had a very slight touch of poisoning.

Discussion: Wild or black cherry (*Prunus serotina* Ehrh) is a tree with reddish-brown branches, which is commonly found in woodlands. The leaves are sharply serrate and the white flowers are small. Blooming occurs in June and July in our vicinity. The ripe fruit is purplish black and small.

*Received for publication, November 9, 1938.

As this happened shortly after vaccination, it made it doubly hard for me and I consider we were fortunate in getting at the cause of the trouble as quickly as we did, thus checking the loss. Of vast importance is the fact that I retained a satisfied client.

CARCINOMA OF THE TONGUE IN A HORSE*

By W. R. HENSON, *Shelby, Ohio*

On June 6, 1938, I was called to the farm of a prosperous client to find the reason why a particular horse did not eat well, the owner believing the teeth to be at fault.

History: The animal presented was a nine-year-old, dark bay gelding. The owner stated that for about two weeks the horse had not masticated his food well, but did not experience any difficulty in swallowing. His appetite was good, but he could not handle hay as well as green forage and grain.

Symptoms: Using "Dr. Hiner's" Indiana spectrum, I made a careful examination with the hand and discovered a pit-like area on the posterior third of the surface of the tongue. The teeth were as nearly perfect as possible.

At this time, the area was as large as an ordinary Petri dish, with very hard edges. This pit-like erosion seemed to have a firm base, was nonpainful, and emitted a disagreeable odor. A light revealed this eroded area to be dark red, with the edges brown. The lymph-glands draining this area were quite hard, but not greatly enlarged.

Clinical diagnosis: From the examination I concluded that the condition was actinomycosis, although I had experienced only one positive case in the equine genus previous to this.

Treatment: One ounce of sodium iodide dissolved in 500 cc of water was given intravenously. The eroded area was swabbed three times daily with a 1:1,000 potassium permanganate solution. Soft feeds were prescribed.

On the third day, I administered 60 grains of potassium iodide, three times daily, and continued this treatment.

On June 13, the condition was much worse, so I anesthetized the patient with 1½ ounces of chloral hydrate intravenously and proceeded to make a more detailed examination. I then discovered the process to be extending down into the pharynx and along one side of the esophagus.

Hope for recovery was nil, so I destroyed the animal and dissected out the tongue and process. This was embalmed in a 10 per cent solution of formaldehyde.

On June 15, I presented the tongue and process to a group of surgeons and pathologists attending the Ohio State Conference. On casual examination, they believed the process possibly to be actinomycosis, but withheld their final diagnosis until sections had been made. Ten days later, I received the laboratory report from Dr. L. W. Goss, which is as follows:

> Tongue greatly enlarged and quite firm, showing diffuse infiltration of tissue. Microscopic examination showed the presence of marked infiltration with stratified, squamous epithelium.

Diagnosis: Squamous-celled carcinoma of the tongue.

*Received for publication, November 21, 1938.

SPONDYLITIS IN THE BOVINE*

By E. R. FRANK, *Manhattan, Kan.*
Kansas State College, Division of Veterinary Medicine

Spondylitis is one form of polyarthritis in which the lesions are located in the articulations of the vertebrae. There is very little on the subject to be found in the literature. Two articles by Dr. C. J. Marshall,[1, 2] which appeared in the University of Pennsylvania Bulletin, contain about the only available information.

It has been suggested that *Brucella abortus* may be the causative organism. However, all the cases examined have been negative to the agglutination test. Probably this condition has often been diagnosed as rheumatism or lumbago.

*Received for publication, December 1, 1938.

The symptoms are very characteristic and constant. The accompanying photograph (fig. 1) of an Ayrshire cow shows the outstanding symptom. The muscles of the back and hind leg become rigid and remain in this position for a few seconds and then the leg is jerked forward. This continues for the first few steps that the cow takes and then the gait becomes almost

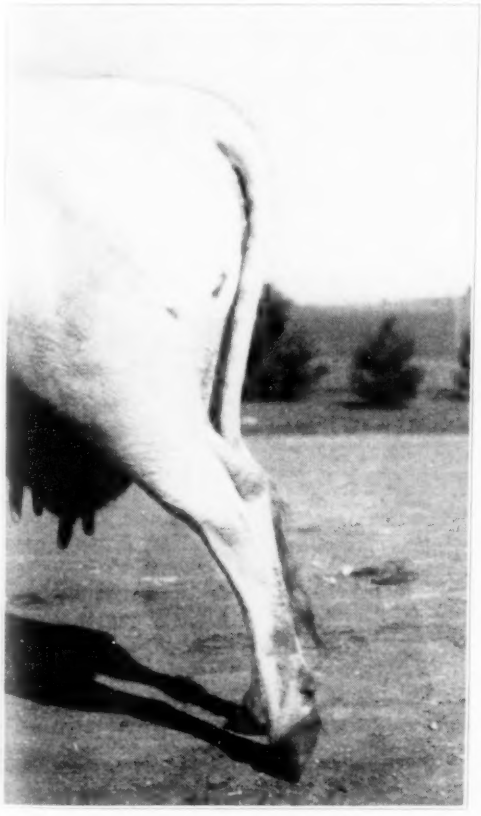

FIG. 1. Ayrshire cow showing characteristic symptom.

normal. The symptoms are most pronounced when the cow first gets up, after she has been lying down for a few hours. Also, they are more pronounced on a cold morning or if she is confined in a stanchion. The symptoms have been noticed for about a year and have remained rather constant.

One Holstein cow was affected very severely in one hind leg and would show the characteristic symptoms for several minutes after getting up, especially if she was confined in a stanchion. Some cows will exhibit the symptoms when they are made to back out of the stanchion or are pushed from one side to the other.

It was possible to purchase a valuable Holstein bull in one of the northern states because it was said that "the extreme cold weather made his rheumatism worse." Both hind legs showed the characteristic symptoms to a marked degree. First, one would be extended rigidly backwards and then the other. He would continue to do this for several minutes without taking a forward step. However, he was able to serve a cow without any noticeable difficulty.

During the course of a year, the symptoms seemed to increase in severity and would last longer. Also, toward the end of the year, it was apparently more difficult for him to get up. At the end of the year, he was completely paralyzed in the posterior limbs.

Postmortem examination revealed a proliferative periostitis involving the affected articulation, which was between the last thoracic and first lumbar vertebrae. The exostosis involving the articulation was extremely large, producing pressure on the spinal cord.

The prognosis in these cases is always guarded. Due to the location of the affected area, it is impossible to determine the extent of the pathological changes that have taken place. However, if the symptoms are of unusual severity, an unfavorable prognosis might be indicated.

Treatment has not been attempted in any of the cases. With the exception of the bull and the cow now under observation, the affected animals have made gradual but slow recoveries.

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SUPERFETATION IN A SOW*

By D. M. SWINEHART, *Elida, Ohio*

The sow, Gold Leaf Connie the Second, which is the subject of this report, is a purebred Chester White, belonging to Mr. D. C. Gamble, of Lima, Ohio, a breeder of

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national reputation. It is indeed fortunate that this unusual event occurred on the farm of a breeder of purebred swine, for the reason that we have access to very carefully kept records of breeding dates.

FIG. 1 (above). Dr. Swinehart (left) holding two of the pigs born Sept. 27, and D. C. Gamble (right) with two of the pigs farrowed Sept. 2.

FIG. 2 (center). Both litters together.

FIG. 3 (below). Dam with litter of eight born Sept. 2, to be followed by a litter of nine born Sept. 27.

The sow was bred first on May 11, 1938. On June 5, she showed signs of estrum and was again mated to the same sire. On September 2, she farrowed eight pigs, and the owner actually thought that the breeding of June 5 was a false alarm. About ten days following the farrowing of September 2, the owner noticed a peculiar sagging of the abdominal muscles of the sow, and he concluded in his own mind that she had ruptured herself in the process of farrowing on September 2.

On the night of September 27, the owner called me and said that he had a sick sow. In answer to questions which I asked him, he advised me that the sow seemed to be nauseated and that she had farrowed on September 2. I proceeded to the farm and found that what the owner had thought were attempts at vomiting were nothing more than labor, preceding farrowing. On my arrival, I found that she had given birth to four normal, healthy pigs. Following my arrival, she gave birth to five more. It is interesting to note that the same interval, namely, 25 days, elapsed between the breeding dates, May 11 and June 5, and the farrowing dates, September 2 and September 27.

This is the second time that Gold Leaf Connie the Second has farrowed. On the first occasion, in the spring of 1938, she produced a litter of ten pigs.

SWINE-POX*

By JOHN B. BRYANT, *Mount Vernon, Iowa*

Swine-pox has been unusually prevalent in eastern Iowa this season. Reports are being received of serious postvaccination losses in pox-affected herds. There must be quite a variation in the severity of pox attacks, or it may be that pox attacks render pigs susceptible to dormant infections and that the secondary invaders are responsible both for the degree of sickness in pox cases and the difficulties encountered when pox herds receive serum and virus.

*Received for publication, December 5, 1938.

To differentiate pox and cholera in the field is not a simple matter. Both are virus diseases and may attack a herd simultaneously. Pox runs a short course in the individual but a more protracted course in the herd and recovery is the rule. The vagaries of the incidence of cholera and its high degree of mortality do not permit of temporizing with highly suspicious cases. On the other hand, if pox and serum-virus reactions are dangerous combinations, then, indeed, differentiation of cholera and pox becomes important.

At any rate, swine-pox is worthy of much study and, in the meagerness of American literature on the subject, it behooves practitioners to make careful notations of all factors observed in pox herds.

Hog lice are commonly observed in pox cases and should be controlled, as it is reasonable to believe that lice may be a factor in the spread of the pox virus. The writer has observed his most severe cases in young pigs. Age seems to be a limiting factor. Pigs closely confined in poorly lighted quarters seem to suffer more severe attacks. A recent case was observed in which the eyes of the older hogs of the herd were severely affected. The lids appeared eroded and swelled until they were almost closed.

Practitioners are keenly anxious for information on swine-pox.

EMBRYOMA OF TESTICLE OF DOG*

By A. A. FEIST, *Saint Paul, Minn.*

Subject: Hound, male, nine years old.

History: Had shown severe pain in the region of the groin.

Symptoms: Examination of the scrotal sac showed only one testicle descended. Painful enlargement in the inguinal area was evident. At first, I thought that this was possibly the inguinal gland but, on second thought, in view of the fact that the patient had only one testicle descended, I decided that the enlargement must be the other testicle and, therefore, advised its removal.

*Received for publication, December 12, 1938.

FIG. 1. Embryoma of testicle of dog.

Histological examination: The tumor (fig. 1) was submitted to Dr. R. Fenstermacher, in charge of the Diagnosis Laboratory, at University Farm, who made the following report:

> The major portion of this tumor is an embryoma. There are areas within this tumor which probably are the result of a rhabdomyoma. The major portion of the tumor is an embryoma. As the name suggests, this is a proliferation of the seminiferous tubules.
>
> This type of tumor, as a rule, is nonmalignant. Very little is known of the prognosis of the same in dogs, but I am informed that in humans it usually results in death within a five-year period. Probably 85 per cent of the human cases terminate fatally.

Following the removal of the testicle, the hound made a complete recovery.

RUPTURED OVIDUCT IN A HEN*

By W. C. SCHOFIELD, *University, La.*
Department of Veterinary Science, Louisiana State University

Subject: Rhode Island Red hen; age, two years.

*Received for publication, December 19, 1938.

History: This was one of two hens sent to the laboratory several weeks ago and was from a flock that had been suffering a few losses. The other bird was autopsied. No indications could be found for autopsy of this bird. The owner was so advised.

Symptoms: The bird was kept in a laying-battery and casual daily observations did not reveal symptoms of any kind. She had been in good production since arrival and the appetite remained good.

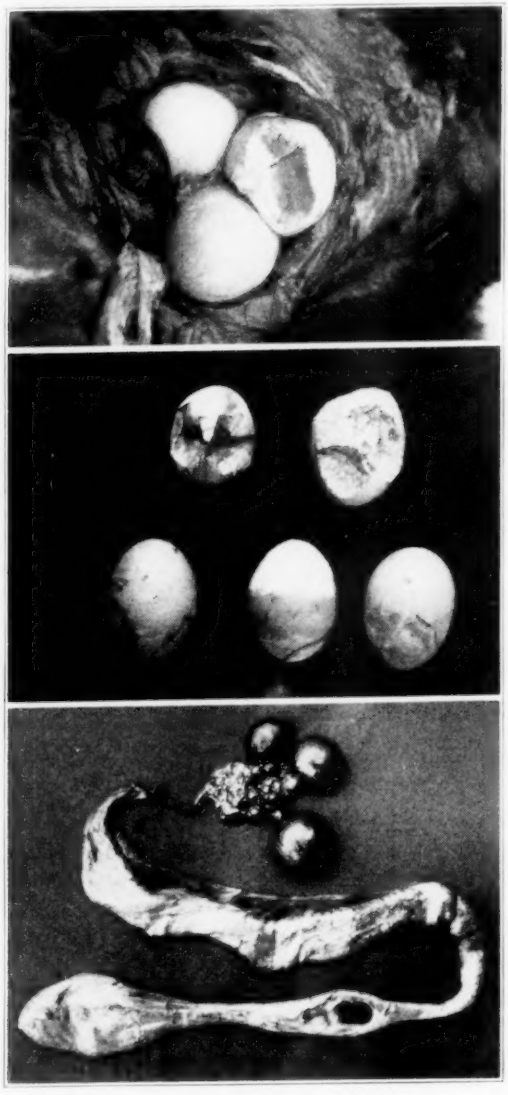

FIG. 1 (above). The three fully formed eggs exposed at autopsy.

FIG. 2 (center). The five eggs found in the abdominal cavity.

FIG. 3 (below). The ruptured oviduct.

Termination: Found dead in battery, on morning of September 13, 1938.

Postmortem findings: On opening the abdominal cavity, there were three fully formed eggs exposed to view (fig. 1). Upon removal of these eggs, two more fully developed eggs were found crowded anterodorsally to the original three. Thus, five fully formed eggs had been laid in the abdominal cavity.

Increased abdominal pressure on the eggs was responsible for cracking two of the five eggs (fig. 2), and this allowed some yolk and albumen to escape into the abdominal cavity.

Diffuse peritonitis was present, evidently the result of rupture of the oviduct, as shown in figure 3. This opening in the wall of the oviduct was more ragged in appearance than is shown in the picture, but a piece of dark paper was inserted into the lumen of the oviduct in order to bring out more clearly the outline of the rupture which was present.

Acknowledgment: The photographs were taken through the courtesy of B. A. Osterberger, assistant entomologist of the Agricultural Experiment Station.

A RANCH OBSERVATION ON FEEDING SHEAF OATS TO LAMBING EWES*

By L. R. VAWTER, *Reno, Nev.*
University of Nevada

In March, 1938, there came to our notice a loss of ewes and lambs in a small ranch flock located in the foothills of the Sierra Nevada Mountains along the Nevada-California border. This loss is of interest from the nutritional standpoint, as it brings out some of the deficiencies of oats when used as the sole article of diet in a manner similar to that reported by Hart, McCollum, Steinbach and Humphrey in 1911.[1] The Wisconsin investigators worked with cattle. Although oats are regarded as

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a valuable feed for ewes when supplemented with other roughage such as timothy or alfalfa hay, we are not aware of any report regarding the oat plant as the sole source of nutriment for pregnant or lambing ewes either under experimental or actual ranch conditions.

On account of deep snow on the ground for three months, a band of about 100 pregnant ewes was confined in large pens with shed shelter from late in December until early in March, subsisting entirely on a diet of sheaf oats. Lambing began about January 15 and continued until early in March. Ewes began to die about the middle of February. Death was preceded by prostration and coma. During a period of about one month, 15 ewes died soon after lambing with a symptom complex suggesting pregnancy disease, vitamin-A and calcium deficiency. The weather was not unusually cold at any time, but sanitary conditions were poor. The lambs were weak when born and at least 15 died within the first three weeks after birth. Snow conditions prevented either reaching the ranch or bringing out any animals for examination until early in March. One lamb was examined and found to have marked lesions of necrophorus infection. Two other lambs, examined postmortem, had limited pneumonic foci in the lungs, but these were not sufficiently extensive to be regarded as the primary cause of death.

One Merino ewe, examined by autopsy after death, was quite emaciated, but the only gross pathologic lesion found was a greatly atrophied liver. This organ was about one-third the normal size for a ewe of its weight and age. The owner stated that some ewes had an eye discharge and appeared to go blind before death. The locality where this ranch is situated has been known for several years to be a phosphorus-deficient area from chemical analysis of forage samples. It is also a borderline area in regard to iodine deficiency. Osteomalacia, with spontaneous bone fractures, has been repeatedly observed clinically in dairy cows and, recently, in Hereford calves on adjacent ranches.

In the instance referred to, a group of ewes was confined and required to exist solely on sheaf oats for a period of approximately three months. Oats are regarded as low not only in calcium but also low in or entirely devoid of vitamin A, and these deficiencies appeared to be exaggerated in the presence of a known existing phosphorus deficiency in the locality. This incident under actual ranch conditions appears to illustrate that the oat plant is inadequate when used as a sole source of nutriment for pregnant or lambing ewes in a manner similar to that observed under experimental conditions with cattle. The losses stopped quite abruptly upon removal of the flock to a larger, clean pen and the addition of a good grade of alfalfa hay to the diet of sheaf oats.

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THE TREATMENT OF PARTURIENT PARESIS*

By A. J. DAMMAN, *Enumclaw, Wash.*

On page 263 of the October, 1938, number of the JOURNAL, there is an article on "Parturient Paresis," by J. W. Kummer. I read the article with a great deal of interest and was pleased to see that the author took the layman to task for the way the animal had been treated. But, on completing the article, I was staggered to read the method by which Dr. Kummer outlined his treatment in this day and age. The air treatment is passé. Let us veterinarians use the more up-to-date method—intravenous injection of calcium gluconate—and the layman will not bother with the treatment of parturient paresis.

We veterinarians in the West have had very good success with this treatment and avoid any udder trouble, as stated in Dr. Kummer's article.

*Received for publication, October 6, 1938.

Yes suh! We'll be seein' yo' all in Memphis, August 28-29-30-31-September 1.

VETERINARY OBSTETRICS (Equine and Bovine). Franz Benesch, Professor of Obstetrics and Director of the Obstetrical Clinic in the Veterinary High School, Vienna. Translated by John G. Wright, F.R.C.V.S., Professor of Surgery at the Royal Veterinary College, London. 258 pages, with 135 illustrations. William Wood and Company, Baltimore, 1938. Price, $4.75.

One recalls Professor Benesch as the clinician who introduced epidural anesthesia in this country. During a sojourn at the New York State Veterinary College, while visiting the United States in 1926, he demonstrated epidural anesthesia at the College and, later, before an audience of the American Veterinary Medical Association, at Lexington, Ky. From that point of departure, the injection of anesthetic solution into the sacro-caudal region rapidly came into general use, not only in cows but also in horses and, to a lesser extent, in swine and pet animals.

A book on obstetrics by this author is therefore a welcome addition to the literature. We are particularly impressed with the author's philosophy, as revealed in the last sentence of his preface, which reads, "An operative interference executed skillfully, according to a plan based upon a careful consideration of the individual requirements, discloses the systematically schooled, scientific veterinarian." This sentence clearly exposes a fact uppermost in mind among capable surgeons and obstetricians. It distinguishes the author as the capable clinician he is known to be.

The warning in the opening chapter to avoid the use of force in effecting deliveries, on the ground that parturition is an elastic process, is a commendable introduction to a work on obstetrics. The author who introduced Phygesen's embryotome in this country naturally recommends an early resort to embryotomy in borderline cases in the cow and mare.

Although the chapter on epidural anesthesia contains but five pages, it is by far the most edifying description of that procedure we have had the opportunity to read. The illustrations of this chapter are graphic, some of them in color. The anesthetic recommended is procaine in a 1 per cent solution, which is 25 per cent stronger than the author recommended ten years ago. The object of epidural anesthesia in obstetrical work is to overcome the objectionable straining and the necessity of avoiding force. The site of the injection in the cow and the mare is nicely portrayed. The simplest type of obstetrical equipment is recommended and the use well described.

The advantages of surgical cleanliness, too often neglected in textbooks on obstetrics and in practice, are set down as immutable laws to successful obstetrics.

The instrumentation recommended for repositioning the malpresented fetus does not follow the antiquated methods of the past but lines of mechanics that seem to bring this phase of obstetrical work up to date.

The chapters on embryotomy, which take up the last seventy-three pages of the book, introduce an entirely new idea, illustrating that phase of obstetrical work. Each illustration has a fine insert of the part of the skeleton sectioned. This style of illustrating gives the operator a clear understanding of what is expected of the instruments he uses, sectioning parts of the fetal carcass in order to render possible its delivery.

The translation seems to have been executed well.

While the more capable bovine and equine obstetricians may find some flaws in

the text, we have been unable to do so. The book is therefore highly recommended to English readers. L. A. M.

ANIMAL PATHOLOGY. Russell A. Runnells, Associate Professor of Veterinary Pathology, Iowa State College, 464 pages, with 127 illustrations. Collegiate Press, Inc., Ames, Iowa, 1938.

This book consists of three parts. Part I deals with general pathology, part II with systemic pathology, and part III with the special pathology of the specific infectious diseases of animals. The second and third parts of the book consist of the subject matter included in the author's previous book, "A Guide to the Study of Special Veterinary Pathology," revised and made more complete.

The text is printed on an excellent quality of paper and the subject matter is well illustrated. There are 127 half-tone illustrations containing approximately 175 figures, the majority of which are of excellent quality and all illustrate material from animal sources. A feature that adds greatly to the usefulness of the book is the inclusion of a bibliography at the end of each chapter.

I am sure that all teachers and students of veterinary pathology will appreciate the publication of this text. As a teacher for more than a quarter of a century, I have felt the need of an English textbook of veterinary pathology. Until now, no American author has had the temerity to undertake the task of writing a complete text. This has not been due to the lack of capable American authors but to the magnitude of the task and the knowledge that there could be no financial reward for the undertaking.

The amount of time and effort required to read the publications of progress made in the study of animal diseases within the last decade and the summarizing and organizing of this material into the form of a textbook constitute a tremendously big job. Furthermore, the size and nature of the text and the necessity for frequent revision in order that such a book may be of a high quality entail such an expense of publication that its cost could not be met by the limited demand without some kind of subsidy.

The author is to be congratulated on this contribution to American veterinary literature. The quality of this work demonstrates that he has made an extensive study of the world's veterinary literature of the past decade or two and is quite capable of the task he has undertaken.

The most severe criticism of the work that I would make is its brevity. Obviously, this shortening of the book is the result of the author's attempt to keep the size of the book and the cost within an amount that would make possible its publication. The discussions on morbid anatomy throughout the text are perhaps as exhaustive as they should be but, in my opinion, a more detailed discussion of the processes leading up to the morbid anatomical states discussed in part I and a fuller discussion of the functional disturbances resulting from the morbid anatomical states discussed in the second part would make the book more useful and interesting. It is my opinion that, with these added features, the book could be made at least 50 per cent larger and the price at least 50 per cent higher without lessening the demand for the book.

There are a great many controversial subjects in pathology, as in all other sciences, and it is to be expected that there will not be complete agreement with the author in a number of instances. To illustrate a few such instances, the following are mentioned:

On page 90, I believe that a distinction should be made between obstructive and toxic jaundice, not because of any practicable importance in veterinary pathology but for scientific reasons. On page 115, it is stated that proliferative changes are not a part of the defense reaction. Is this true? On the same page, it is stated that the proliferative changes concern themselves with the connective tissue cells. This is true, but is it the whole truth?

On page 122, it is stated that a purulent exudate is the type associated with suppuration. True again, but may there not be a purulent exudate (as generally accepted) without suppuration? On page 145, it is stated that vitamin D is abun-

dant in green feed. Is this true? In the discussion of the pathology of rickets on page 147, no mention is made of the excessive production of osteoid tissue. Is not this the most characteristic feature of that condition?

On page 231, too little emphasis is placed on acute bronchiolitis as a common cause of atelectasis. Acute bronchiolitis is not mentioned as a cause of acute alveolar emphysema. That apparently is the most common cause of this lesion. A rather detailed discussion on lobar pneumonia is given on pages 238, 239 and 240, while only two paragraphs are devoted to a discussion of broncho (lobular) pneumonia. Is there any assurance that the condition described as lobar pneumonia in man ever occurs in the lower animals? It is true that in pneumonia in the lower animals the lobe, or several lobes, may be, and frequently are, involved in their entirety, but the condition differs from the description of lobar pneumonia in man both clinically and pathologically.

On page 289, alcohol is listed as a cause of cirrhosis and, on page 352, as a cause of neuritis. Is not the possibility of alcohol causing either condition in animals so remote that its mention might be excluded? On page 302, it is stated that injury to the glomerulus sympathetically affects the tubule. A fuller discussion of the physiological relationships of these units of the nephron and the reasons for the associated lesions would be of more interest and value.

Some of the citations above concern controversial subjects, while others illustrate instances of too much brevity. However, there is no other veterinary pathology in English that is equal to this book in quality and thoroughness. It is a commendable contribution to our professional literature and warrants the support of all students of, and workers in, animal diseases. It is to be hoped that the author will make it his major professional objective to enlarge and keep the work up-to-date by frequent revisions. It is also hoped that the institution which he serves and the veterinary profession will make it possible for him to attain this objective. E. T. H.

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Cardiovascular and Other Lesions in Calves Fed Diets Low in Magnesium. L. A. Moore, E. T. Hallman and L. B. Sholl. Reprint from *Arch. Path.*, xxvi (1938), pp. 820-838. Illus.

Bovine Mastitis. III. A Comparison of the Bacteriological and Physiological Reactions of Normal and Mastitis Milk from Young Cows. Ralph B. Little. Reprint from *Corn. Vet.*, xxviii (1938), 1, pp. 23-33.

THE EFFECT OF CERTAIN BACTERIAL TOXINS UPON SOME RESPIRATORY MECHANISMS OF ANIMAL TISSUES. Walter Reginald Wooldridge and Constance Higginbottom. Biochem. Jour., xxxii (1938), p. 1718.

The toxins obtained from *Clostridium welchii*, types A, C and D, inhibit the aerobic oxidation of succinate by aqueous suspensions of minced small intestine. This inhibition is reduced to a considerable extent by the presence of antisera which neutralize the *a* toxin of *C. welchii*. The inhibitory action of the toxins is greatly reduced by heating for 15 minutes at 80 degrees. The inhibitory action appears to be due to the *a* toxin. The toxins of *C. edematiens*, *C. septique* and *C. tetani* exert no action different from that induced by a precipitate prepared similarly from the broth medium. This inhibitory action is not due to an effect upon either the succinic dehydrogenase itself or the indophenol oxidase, but may be due to an action upon some intermediate carrier catalyst at present unidentified. The pathogenicity of *C. welchii* may be associated in part with this inhibitory action of its *a* toxin upon an important respiratory mechanism of tissues.

THE INFLUENCE OF COBALT ON PINE DISEASE IN SHEEP. Harold Hartman Corner and Alexander Martin Smith. Biochem. Jour., xxxii (1938), p. 1800.

Pine disease in sheep in the Cheviot region of Scotland is a nutritional anemia which can be cured and prevented by the administration of cobalt in the form of cobalt chloride. The disease is not due to a deficiency of iron, copper or manganese. The beneficial results previously obtained from the feeding of iron compounds may be attributed to the presence of cobalt with iron compounds used. The administration of 1 mg of cobalt per day for 14 days is sufficient to prevent the disease on severe pining land for a period of six months. A similar quantity is effective as a cure.

NICOTINIC ACID IN THE PREVENTION OF BLACKTONGUE IN DOGS. W. H. Sebrell, R. H. Onstott, H. F. Fraser and F. S. Daft. Jour. Nutri., xvi (1938), p. 355.

Recession of acute symptoms of blacktongue followed the administration of either sodium nicotinate or nicotinic acid. The total quantity of nicotinic acid necessary to cause the acute symptoms of blacktongue to subside appears to be from 20 to 60 mg. The wide variation in the amount of nicotinic acid required to prevent recurrence of acute symptoms of blacktongue suggests that some factor other than body weight may be involved. A semi-weekly dose of 3.0 mg of nicotinic acid appears to be barely sufficient to prevent blacktongue under the conditions of these experiments. A semi-weekly dose of 10.0 mg of nicotinic acid was ample to prevent blacktongue for a period of six months.

OXIDATIVE CHANGES INDUCED BY A MINERAL-DEFICIENT DIET. Max Kriss and Arthur A. Smith. Jour. Nutri., xvi (1938), p. 385.

The respiratory quotients of rats receiving a diet deficient in inorganic salts were almost identical with those of control animals during the first month of the diets. During the second and third months, however, the respiratory quotients of the low-salt animals were significantly lower than those of their controls. The mineral deficiency apparently resulted in the oxidation of relatively larger proportions of fat and smaller proportions of carbohydrate, while it was practically without effect on the protein metabolized. In spite of the differ-

ences in the total heat production, there were no significant differences between the specific dynamic effects of the diets compared, as these effects are generally defined. This is explained by the fact that the increases in total metabolism caused by the mineral deficiency may be fully accounted for by similar increases in the basal metabolism.

THE HISTOPATHOLOGY OF NEUROMALACIA AND "CURLED TOE" PARALYSIS IN THE CHICK FED LOW RIBOFLAVIN DIETS. Paul H. Phillips and R. W. Engel. Jour. Nutri., xvi (1938), p. 451.

A low riboflavin diet may cause two types of a nutritional deficiency: (1) a rapidly acute paralysis characterized as neuromalacia and (2) a more slowly developing form which causes curled toe paralysis in the growing chick. These symptoms may be prevented by the addition of crystalline riboflavin to the basal diet. In the acute form hypertrophy and change in appearance of the peripheral nerve-trunks can be readily observed on gross inspection. Degenerative changes appear in the myelin of the nerves accompanied by Schwann cell proliferation and axis cylinder swelling and fragmentation. Histologic changes also occurred in the spinal cord. Neuromuscular end plate degeneration and muscle fiber degeneration frequently accompanied the paralysis. A 6 per cent level of oat grass was marginal in its flavin content as determined by microscopic examination of the sciatic nerve. It was found that addition of the anti-dermatitis factor to the low flavin basal diet considerably reduced the histopathologic changes of the spinal cord. It is concluded that riboflavin is necessary in the growing chick for the normal function and maintenance of the nervous system and is specifically associated with myelin metabolism.

OBSERVATIONS ON THE LIFE HISTORY OF RAILLIETINA ECHINOBOTHRIDA AND OF RAILLIETINA TETRAGONA (CESTODA). Margery W. Horsfall. Jour. Parasitol., xxiv (1938), p. 409.

Two species of ants, *Tetramorium caespitum* and *Pheidole vinelandica*, have been demonstrated as intermediate hosts for *Raillietina echinobothrida* and *R. tetragona* in the United States. Under natural outdoor conditions, the cestodes were present throughout the year in chickens. The ants were active from March 15 through December, but the ants were found naturally infected only from approximately June 1 through December, in both 1935 and 1936. Both cysticercoids and adults of *R. echinobothrida* and *R. tetragona* may be distinguished only by the number of rows of hooks, the number of hooks and the size of the hooks in the rostellar crown. These two species of cestodes are found generally along the Atlantic Coast and Gulf of Mexico and in the Mississippi Valley. The species of ants serving as intermediate hosts are more widely distributed than the cestodes. While the observations on the life histories of these cestodes do not include the developing stages of the cysticercoids in the ant, they do tend to limit the time and place of this development to the ant nest.

OBSERVATIONS ON THE BIOLOGY OF THE POULTRY CESTODE, DAVAINEA PROGLOTTINA, IN THE INTESTINE OF THE HOST. P. P. Levine. Jour. Parasitol., xxiv (1938), p. 403.

The author describes a new method for counting the small tapeworm, *Davainea proglottina*, in the intestines of the chicken. During the summer months, the peak of the segment discharge occurred between 5 p. m. and 9 a. m.; in the winter, the peak was reached between 2 p. m. and 4 p. m. This shift in the time of segment discharge is apparently dependent on the shortening of the days in the winter, with the resultant effect that the feeding time of the birds is altered. The time of greatest segment discharge is apparently dependent on the shortening of the days in the winter, with the resultant effect that the feeding time of the birds is altered. The time of greatest segment discharge may be altered at will by changing the feeding hours of the host.

The total number of segments discharged each day tended to remain at about the

same level as long as feeding and other environmental influences were unchanged under optimal conditions. As far as the parasite is concerned, each worm is capable of shedding one ripe segment about every 24 hours. Feeding the host on rations with little or no food value not only resulted in a marked decrease in segment production but in some instances caused segment production to cease entirely. The starvation of the host for 24 hours caused a decrease in the number of segments discharged during the following week, after which there was a return to normal discharge.

SPECIES DIFFERENTIATION IN THE COCCIDIA FROM THE DOMESTIC SHEEP. John F. Christensen. Jour. Parasitol., xxiv (1938), p. 453.

Results of a survey of fecal samples from 100 sheep for coccidial oöcysts showed 96 per cent infection, 34 per cent being pure infections with a single species and 62 per cent being mixed infections with two to four species each. Size, shape, color, sporulation time and morphology of the unsporulated oöcyst were the criteria used for species identification. The oöcysts of *Eimeria parva, E. nina-kohl-yakimovi, E. faurei, E. arloingi* and *E. intricata* are redescribed. Oöcysts conforming to the description of those of *E. galouzoi* were found in the spherical range of *E. parva*, and oöcysts similar to those described for *E. aemula* were observed among atypical and capless oöcysts of *E. arloingi*. Two new species, *E. pallida* and *E. granulosa*, are described from oöcysts found repeatedly in fecal samples.

AVIAN TUBERCLE BACILLI IN THE UDDER OF A GOAT. Change of S to R forms. A. Stanley Griffith. Jour. Comp. Path. and Ther., li (1938), p. 151.

A strain of avian tubercle bacilli which had localized in the right half of the udder of a goat, soon after the subcutaneous injection of a culture, persisted there for a period of about nine years and was grown on successive occasions from the udder secretions. During the residence of the strain in the udder, there was a change from the S to the R form of the tubercle bacillus. The R form first appeared as an admixture in a culture made 8½ years after the injection of the goat and was the only form in the last positive culture obtained eight months later. The R form differed from the S form in cultural characteristics and in agglutinability and agglutinin absorption. No differences in virulence between the two forms were shown in experiments on fowls, rabbits and guinea pigs. The goat died eleven years after injection. The right half of the udder was atrophied and showed no sign of tuberculosis either macroscopically or microscopically.

SEASONAL HYPOMAGNESAEMIA OF THE BOVINE WITHOUT CLINICAL SYMPTOMS. W. M. Allcroft and H. H. Green. Jour. Comp. Path. and Ther., li (1938), p. 176.

Eighty-nine Hereford cows on pasture during the entire year on a Kent farm without shelter and with hay only as a supplementary feed in winter, apart from access to a commercial mineral lick, showed a regular seasonal variation in serum magnesium from a maximum in August, 1937, to a minimum in December, with a return in February, 1938, almost to the June, 1937, level. Winter levels were frequently less than one-half the summer levels, but no clinical symptoms of tetany or signs of nervous hyperexcitability were shown. A smaller group of 15 cows of mixed breed in an adjoining pasture on the same farm did not show such pronounced variations. The addition of magnesium oxide to the mineral mixture appeared to alleviate, to a limited extent, the acuteness of the fall in serum-magnesium level from October to December. The pasture was not considered deficient in magnesium content. The authors offer no explanation for the variations.

THE RESISTANCE OF THE SWINE FEVER VIRUS TO PHYSICAL AGENCIES AND CHEMICAL DISINFECTANTS. George Slavin. Jour. Comp. Path. and Ther., li (1938), p. 213.

The virus of swine fever is very resistant to changes in hydrogen-ion concentration. Infected brick and hay were exposed to atmospheric influences. As other workers have found, the virus survived only a few days. In association with these experiments, pigs were exposed to infection by feeding virus, by dropping virus into the eyes and by injecting virus into the nasal cavities. Although all of the pigs were fully susceptible to swine fever, none of them developed the disease until they were later inoculated subcutaneously with virus to prove their susceptibility. These results confirm the work of other workers and explain the irregular results of contact experiments. The resistance of the virus to chemical disinfection was investigated. The virus was destroyed in 15 minutes by a 5 per cent solution of phenol and by a solution of hypochlorite containing 1.66 per cent available chlorine. The hypochlorite is preferred on account of its cleansing and deodorant action; its action, however, is less persistent than that of phenol.

THE EFFECT OF ETHER ANESTHESIA ON THE VOLUME OF PLASMA AND EXTRACELLULAR FLUID. Ferdinand F. McAllister. Amer. Jour. Physiol., cxxiv (1938), p. 391.

The effect of ether anesthesia on the plasma volume of dogs was measured. In 23 experiments, one hour of anesthesia caused an average decrease in plasma volume of 11.0 per cent. When calculated from the rise in concentration of the serum proteins, the fluid loss averaged 9.3 per cent. The hematocrit rise was almost double that which could be accounted for by the plasma volume reduction. This discrepancy was less pronounced after splenectomy. Within four hours after the removal of ether, the plasma volume returned to the preanesthesia level and in some cases it rose well above that level. The decrease of plasma volume was independent of changes in body temperature.

INVITATIONS FOR 1941

(*Continued from page* 113)

ern states, and the other the New England and Middle Atlantic states.

Present plans call for holding the 1941 meeting in the tentatively designated central zone or district, and the purpose of this announcement is to call for invitations from cities located in the 14 states above named. These invitations should be on file with the Secretary of the A. V. M. A. not later than April 1, 1939, so that ample opportunity may be provided for an inspection of the cities extending these invitations some time between April 1 and the Memphis meeting, the latter part of August. A committee specially appointed for the purpose will make an investigation of the convention facilities offered by these cities, and their report will be made to the Executive Board at Memphis.

PUERTO RICO AND VIRGIN ISLANDS ACCREDITED

Puerto Rico and the Virgin Islands are now practically free of bovine tuberculosis and have been officially designated as modified accredited areas by the U. S. Department of Agriculture. The tuberculin testing in Puerto Rico covered the 77 municipalities of the island and involved more than a quarter of a million cattle. In the Virgin Islands, some 12,000 cattle were tested without a single reactor being disclosed.

California still has ten counties in which the extent of bovine tuberculosis has not been reduced to less than 0.5 per cent, although the work in these counties is progressing and will be stepped up during the next few months, with a view to getting the Golden State on the accredited list before the end of the present calendar year.

He that judges, without informing himself to the utmost that he is capable, cannot acquit himself of judging amiss.—*Locke.*

ARMY VETERINARY SERVICE

Regular Army

Lt. Colonel Joseph F. Crosby, now on temporary duty at headquarters, First Corps Area, Boston, Mass., from Fort Devens, Mass., is relieved from assignment and station at Fort Devens, effective upon completion of his temporary duty, on or about April 15, 1939, and assigned to the office of the Surgeon General, Washington, D. C., for duty.

The appointment of Captain Frank Arnold Todd, Veterinary Corps Reserve, as first lieutenant in the Veterinary Corps, Regular Army, with rank from December 1, 1938, and his assignment to station at Fort Devens, Mass., are announced.

The promotion of 1st Lieut. Walter T. Carll, to the grade of captain, with rank from December 4, 1938, is announced.

Lt. Colonel Gardiner B. Jones is assigned to Fort Snelling, Minn., for duty effective upon completion of his tour of foreign service in the Philippine Department.

Lt. Colonel Daniel H. Mallan is assigned to Langley Field, Va., for duty effective upon completion of his tour of foreign service in the Panama Canal Department.

Paragraph 5, S.O. 225, W.D., 1938, which relieves Lt. Colonel Jean R. Underwood from his present assignment and duty at the Army Medical Center, Washington, D. C., and directs him to sail on or about February 7, 1939, for the Philippine Department, is revoked.

Lt. Colonel Isaac O. Gladish is relieved from duty at Fort Sam Houston, Texas, effective in time for him to proceed to San Francisco, Calif., and sail on transport scheduled to leave that port on or about February 28, 1939, for the Philippine Department; upon arrival will report to the commanding general for assignment to duty with the Veterinary Corps.

Colonel George H. Koon is relieved from assignment and duty at headquarters, Eighth Corps Area, Fort Sam Houston, Texas, effective on or about April 1, 1939, is then assigned to headquarters, First Corps Area, Boston, Mass., and will proceed to that station and report to the commanding general, First Corps Area, for duty with the Veterinary Corps. He is also assigned to additional duty at the Boston Quartermaster Depot and as attending veterinarian, Harvard University.

Lt. Colonel Jean R. Underwood is relieved from assignment and duty as instructor and director, Army Veterinary School, Army Medical Center, Washington, D. C., effective on or about March 15, 1939, is then assigned to headquarters, Eighth Corps Area, Fort Sam Houston, Texas, and will proceed to that station and report to the commanding general, Eighth Corps Area, for duty with the Veterinary Corps.

Lt. Colonel Nathan M. Neate is relieved from assignment and duty at Langley Field, Va., effective on or about January 10, 1939, and directed to proceed to Fort Knox, Ky., for duty.

Major Verne C. Hill is relieved from duty at Fort Leavenworth, Kansas, effective in time to proceed to New York, N. Y., and sail on transport scheduled to leave that port on or about February 7, 1939, for the Philippine Department and, upon arrival, will report to the commanding general for assignment to duty with the Veterinary Corps.

Lt. Colonel Philip H. Riedel is relieved from his present assignment and duty at Fort Sam Houston, Texas, is then assigned to duty at the Eighth Corps Area General Depot, Fort Sam Houston, Texas, and will report to the officer in charge for duty. Lt. Colonel Riedel is assigned to additional duty as attending veterinarian, Randolph Field, Texas.

Captain Maurice W. Hale is relieved from his assignment and duty at Fort Sam Houston, Texas, is then assigned to duty at station hospital, Fort Sam Houston, Texas, and will report to the commanding general, Eighth Corps Area, for duty.

Captain John L. Owens is relieved from assignment and duty at Randolph Field, Texas, effective on or about January 15, 1939, is then assigned to the Saint Louis Medical Depot, Saint Louis, Mo., and will proceed to that station and report to the commanding officer for duty.

Lt. Colonel Raymond A. Kelser has been directed to proceed to Fort Robinson, Nebr., and Fort Reno, Okla., on temporary duty, for the purpose of investigating the prevalence of equine strangles and associated conditions and for the formation of plans for the control and prevention of equine encephalomyelitis. While on this temporary duty, Colonel Kelser has been granted twelve days detached service for the purpose of attending, without additional expense to the Government, the University of California Veterinary Conference, to be held at Davis, Calif., January 3-6, 1939, and a meeting of the Intermountain Livestock Sanitary Association at Salt Lake City, Utah, on January 9, 1939.

Lt. Colonel Louis L. Shook is relieved from assignment and duty at Fort Bliss, Texas, effective at such time as will enable him to comply with this order, is then assigned to Fort Hoyle, Md., will proceed at the proper time to San Francisco, Calif., and sail on transport scheduled to leave that port on or about June 20, 1939, for New York, N. Y., and, upon arrival thereat, will proceed to Fort Hoyle, Md., and report for duty.

Captain Richard G. Yule is relieved from assignment and duty at Fort Moultrie, S. C.,

and from additional duty as attending veterinarian, Fort Screven, Ga., effective on or about February 1, 1939, is then assigned to duty at Fort Leavenworth, Kan., will proceed to that station and report to the commanding officer for duty.

Veterinary Corps Reserve

NEW ACCEPTANCES

(First lieutenants)

Bonds, Thomas Edison, Adamsville, Ala.
Burke, Wm. Zebulon, 1322 W. Rosewood, San Antonio, Texas.
Davidson, Wm. Max, 307 E. Holmes St., Huntsville, Ala.
Jacobson, Otho Henry, Public Safety Bldg., Salt Lake City, Utah.
Kendall, Hunter Ernest, 412 State Office Bldg., Columbia, S. C.
McGinnis, Clifford Ladell, 3000 North University, Peoria, Ill.
Stigers, Arthur Leroy, 529 10th St., W., Des Moines, Iowa.

SEPARATIONS

1st Lieut. Robert George Elmore, died September 2, 1938, from lobar pneumonia. Captain Frank Arnold Todd, accepted appointment in Veterinary Corps, Regular Army, December 1, 1938.

PROMOTIONS

To Captain: Rudolph Julius Steffen, Route No. 1, West Broad, Horseheads, N. Y.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Captain Leo A. Hock, Ohio District, Fort Hayes, Ohio.
1st Lieut. Walter A. Anderson, Seattle Q. M. Depot, Seattle, Wash.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY WITH CCC

Captain F. Arnold Todd, Waterbury, Vt. (Accepted appointment in Regular Army.) 1st Lieut. Harold O. Sheetz, Kentucky District. 1st Lieut. Ray V. Schoentrup, Ohio District. 1st Lieut. Joseph D. Manges, Fort Lewis, Wash.

Hoskins Memorial Medal Awarded

Captain Thomas C. Jones, V. C., was awarded the Hoskins Memorial Medal for the year 1938-1939, at the recent graduation exercises of the Army Veterinary School, Army Medical Center, Washington, D. C. Captain Jones (Wash. '35) was first commissioned in the Veterinary Corps of the Regular Army on September 30, 1935, as a first lieutenant, and was promoted to the grade of captain on July 24, 1938.

The Hoskins Memorial Medal, sponsored by the American Veterinary Medical Association, is awarded to the student attaining the highest standing in the Army Veterinary School.

106 Billion Pounds of Milk Used Each Year

More than 100 billion pounds of milk are produced and consumed in the United States each year. Production on farms in the United States in recent years has averaged about 103 billion pounds, according to the Bureau of Agricultural Economics. In addition to this farm-produced milk, about 3 billion pounds are produced in areas not classified as farms near cities and villages.

Of this 106-billion-pound total, more than 30 billion pounds are consumed in cities and villages as fluid milk and cream. This city consumption of milk and of cream, in terms of its milk equivalent, represents nearly a third of the total production each year.

More than half of the total production each year goes into manufactured dairy products, such as butter, cheese, evaporated milk, condensed milk, ice cream, malted milk, and so forth. Butter is the most important manufactured product. Creamery butter production alone takes more than 30 billion pounds of milk each year, or approximately a third of the total annual milk output. Another 10 per cent is used for producing butter on the farm. Cheese accounts for nearly 6 per cent of all milk produced each year, and ice cream for 3 to 4 per cent.

Farm folks consume an additional 12 or 13 billion pounds of milk annually.

The Bureau reports that in 1936, the latest year for which figures are complete, production of milk in the United States was approximately 825 pounds per person.

Bureau Transfers

DR. CLAUDE A. SMITH (O. S. U. '35), from San Juan, Puerto Rico, to Lansing, Mich., on tuberculosis eradication.

DR. CLIFFORD B. BRATAGER (Chi. '17), from Sioux Falls, S. Dak., to Grand Forks, N. Dak., in charge of meat inspection.

DR. M. E. SCHWAB (Cin. '16), from Grand Forks, N. Dak., to Watertown, S. Dak., in charge of meat inspection.

LEGAL OPINIONS

LIABILITY OF VETERINARIAN FOR MALPRACTICE

General Considerations

A veterinarian, in the absence of a special contract, engages to use such reasonable skill, diligence and attention as may be ordinarily expected of persons in that profession; he does not undertake to use the highest degree of skill nor an extraordinary amount of diligence; in other words, the care and diligence required are such as a careful and trustworthy man would be expected to exercise.

B—— vs. *P*——, 45 N. W. 694
C—— vs. *W*——, 65 Am. Dec. 761
W—— vs. *G*——, 71 Me. 21

THIS department of the JOURNAL will be conducted by Mr. Joseph M. Kotz, who has been legal counsel for the American Veterinary Medical Association for the past six years. The purpose of this department is not to offer legal advice in cases involving individual veterinarians, but Mr. Kotz will discuss, each month, actual cases, taken from court records, in which veterinarians have been involved in litigation. Legal questions of general interest to the profession will also be discussed in this column from time to time.

Castrating a Colt

In an action (*P*—— vs. *G*——, 25 Wis. 446) to recover damages for malpractice in castrating a colt, resulting in the death of the animal, it has been held not to be error to instruct the jury: "If the injury was purely an accident, or arose from some other cause, while defendant exercised a proper degree of skill, he was not liable," although there was no positive evidence of such accident or other cause.

Throwing a Horse

In *S*—— vs. *S*——, 167 Ala. 241, plaintiff alleged that the defendant, a veterinarian, had so negligently or unskillfully treated his horse for a spavin that the horse died as a result. It appears from the evidence that there was considerable doubt that the mere application of a cautery to the spavin accounted for the death of the animal a few hours thereafter, but that its death resulted from a rupture of the diaphragm, caused either by violently throwing the horse to the ground or by its struggle to free itself from the bonds which held its feet together during the operation, it appearing that the place selected for throwing the horse—being on the side of a hill, whereas, a level spot was available—was so unsuited to the purpose as to cause the horse to be thrown with unusual and unnecessary violence, thereby causing its injury and death. Since throwing the horse was necessary for such operation, it was held that the manner in which said horse was hobbled and thrown by the defendant definitely showed a lack of skill. Judgment was entered against the veterinarian.

Vaccination of Hogs

In an action (*P*—— vs. *L*——, 204 N. W. 301) against the defendant, a veterinarian, for negligent vaccination of plaintiff's hogs, it appears that plaintiff observed symptoms of illness in a few of his hogs and called the defendant to inspect them. At that time, plaintiff had a drove of 193 hogs; 139 of them had been purchased recently. The sick hogs were part of such purchase. The defendant advised the vaccination of the entire drove on the theory

that the sick hogs were probably, or at least might be, afflicted with cholera. Arrangements were then made for the vaccination on the following day. Within a period of a month from that date, 93 of the hogs died. Plaintiff took the position that none of his hogs had died of cholera but that they all had died of so-called "blood poisoning," and that their condition had been caused by improper vaccination; that the poisoning had been caused by the improper and unclean condition of the needle used by the defendant in injecting the hogs at the time of vaccination. It appeared that hogs in that territory were subject to a half-dozen different forms of swine diseases, all of which were caused by a germ finding its way into the bloodstream, and there was entire absence of evidence of any causal relation between the defendant's alleged negligence and the plaintiff's loss. It was held that the trial court properly directed a verdict for the defendant.

Fine Unlicensed Illinois Practitioner

Frank Kline, rendering works operator, of Streator, Ill., was arrested and fined for practicing veterinary medicine without the formality of procuring a license. The complainant was Inspector Fitzgerald, of the Department of Registration and Education, working for the Illinois State Board of Veterinary Examiners.

With the defendant came a delegation of clients ready to vouch for his qualifications. "Whether licensed or not, he can diagnose a case and tell us how to cure sick live stock," the delegation argued in their protest to the court. They also asked that the defendant be permitted to take a special examination so that he could continue his work.

A survey of the Illinois situation, made in the line of duty by Chief Veterinarian Welch and his successor, Dr. H. C. Rinehart, present incumbent of that office, indicates that the shortage of veterinarians engaged in practice in that state is making it increasingly difficult to convict violators of the Veterinary Practice Act.

Two Ohioans Prosecuted for Illegal Practice

In Canton, Ohio, a fine of $50.00 and costs was imposed recently on each of two violators of the Veterinary Practice Act. The men, Myrth S. Gaskell and C. C. Young, also were ordered to jail for 60 days, but sentence was suspended on condition that they refrain from further violation of the law.

Gaskell was charged with examining chickens and dispensing advice and drugs for treatment and cure of chicken diseases for a fee, without first obtaining a license to do so. Young was charged with practicing surgery on a cow without having the proper license from the state.

These convictions follow in the wake of a vigorous campaign against "quacks," launched by veterinarians of the Buckeye State. Since October, 1937, 20 persons in Ohio have been convicted of illegal practice and eleven others have agreed to cease operations.

Hit-and-Run Driver Injures Dog; Fined

A Philadelphia magistrate recently fined Edward Spiegel $28.50 on a charge of hit-and-run driving, in an accident in which "Buddy," a Spitz dog, was injured. The Pennsylvania Society for Prevention of Cruelty to Animals charged that Spiegel was the driver of the automobile and that he did not stop and "render assistance."

Thou may'st be sure that he that will in private tell thee of thy faults, is thy friend, for he adventures thy dislike, and doth hazard thy hatred; for there are few men that can endure it, every man for the most part delighting in self-praise, which is one of the most universal follies that bewitcheth mankind.—*Sir Walter Raleigh.*

Read not to contradict and confute, nor to believe and take for granted, nor to find talk and discourse—but to weigh and consider.—*Bacon.*

SOUTHERN ALABAMA-WEST FLORIDA VETERINARY MEDICAL CLUB

An impromptu program was carried out as the Southern Alabama-West Florida Veterinary Medical Club held its second semi-annual meeting, in Atmore, Ala., September 15, 1938. By a unanimous vote, it was decided to create a permanent organization for this new society. Accordingly, an election of officers was held, with the following results: President, Dr. A. L. Holloway, of Atmore, Ala., and secretary-treasurer, Dr. E. M. Nighbert, of Cantonment, Fla.

Pink-eye in cattle, jaundice in dogs, and sleeping sickness in horses were the subjects discussed during the day.

Pink-eye, as it affects cattle in certain sections of Alabama, has become a serious problem, it was pointed out, since no new methods for its control have been developed. Sodium chloride rubbed freely into the eye was mentioned as an effective treatment, this method having been used by a cattle-owner with very good results.

Jaundice in dogs, a vector-borne condition, is one of the very important problems which confront small-animal practitioners, it was explained. Much research is necessary before the disease can be controlled and a cure perfected, since, at present, treatments even at the onset of the condition prove unsatisfactory.

The subject of sleeping sickness in horses stimulated a very good discussion and many interesting facts about this disease were brought out.

One of the main features of the meeting was a clinic held at Dr. Holloway's hospital.

Drs. I. S. McAdory and L. E. Starr, of the Alabama Polytechnic Institute, Auburn, were guests of honor. Dr. McAdory talked on the training of students for the veterinary service. He emphasized the fact that high standards for the profession as a whole can be achieved only through correspondingly high standards of education and preparatory training, pointing out the importance of interneship and general practice work in the field. Dr. Starr led the discussions and contributed many valuable viewpoints on technical matters.

E. M. NIGHBERT, *Secretary.*

BATON ROUGE VETERINARY GROUP

The veterinarians of Baton Rouge and vicinity are meeting for organized discussions of subjects chosen by the group. The meetings are held the first Tuesday in each month. The October and November meetings were each attended by 13 veterinarians. There are four Bureau veterinarians regularly stationed in Baton Rouge; five practitioners; two veterinarians with the State Live Stock Sanitary Board; one man in charge of municipal meat inspection, and three veterinarians, a parasitologist and two bacteriologists at the Louisiana State University who form the nucleus for this group.

W. T. O.

NORTHEAST KANSAS VETERINARY MEDICAL SOCIETY

The Northeast Kansas Veterinary Medical Society held a meeting at the Jayhawk Hotel, Topeka, in the afternoon and evening of December 16, 1938.

Round-table discussions on encephalomyelitis and bot treatments and general pointers on practice comprised the afternoon session. In the evening, 34 veterinarians and the ladies attended a dinner served at the hotel. Plans for another meeting, to be held in the spring or early summer, were announced.

R. F. COFFEY, *Chairman.*

ATLANTA VETERINARY MEDICAL SOCIETY

The Atlanta Veterinary Medical Society held its December meeting at the Robert Fulton Hotel, Atlanta, Ga., on December 7, 1938, and entertained the State Board of Veterinary Examiners at a luncheon.

Dr. J. M. Sutton, State Veterinarian, gave a short talk on the standards and ethics of the veterinary profession in Georgia. Other members of the Board discussed what the various practitioners can do in their own localities to increase the prestige of the profession.

Membership in the A. V. M. A. was discussed and all present pledged full coöperation with the national group in securing new members.

CHAS. C. RIFE, *Secretary.*

SOUTH DAKOTA VETERINARY MEDICAL ASSOCIATION

The annual meeting of the South Dakota Veterinary Medical Association was held at the Carpenter Hotel, Sioux Falls, December 15-16, 1938. Dr. F. F. Smith, of Emery, presided and introduced Hon. A. N. Graff, mayor of Sioux Falls, who delivered the address of welcome.

On the program of the first day, Dr. R. Fenstermacher, of the University of Minnesota, Saint Paul, Minn., presented a paper on "Unusual Conditions Found in Live Stock," and Dr. S. H. McNutt, of Iowa State College, Ames, Iowa, spoke on "Vitamin E."

It was only fitting that the 100 members and ladies who attended the banquet in the evening were served "turkey with all the trimmings," since South Dakota is famed the country over for its fine turkeys—a reputation which the veterinary profession of this state has vigilantly guarded. Dr. Fenstermacher officiated as toastmaster and introduced several visitors, who gave impromptu talks. Dr. E. M. Walker, of Lemmon, who attended the Thirteenth International Veterinary Congress, in Switzerland, the past summer, discussed some of the interesting phases of his trip. Immediately after the banquet, a dancing party was held and, for that occasion, commercial houses serving the territory furnished a seven-piece orchestra.

The activities of the second day included a paper given by Mr. Guy S. McDonald, of the South Dakota Agricultural College, on "Feeds and Feeding." Dr. P. V. Neuzil, of Blairstown, Iowa, who has gained widespread recognition for his work in the field of poultry diseases, spoke on "Building a Poultry Practice." The election of officers followed, with these results: President, Dr. L. L. Dunn, of Dell Rapids; vice-president, Dr. J. M. Bailey, of Parker, and secretary-treasurer, Dr. G. E. Melody (reëlected), of Gettysburg.

A round-table discussion of swine diseases, led by Drs. Frank Breed, of Lincoln, Neb., and F. F. Smith, was a feature of the afternoon session. Dr. C. H. Fauks, of Pierre, inspector-in-charge for the U. S. Bureau of Animal Industry, gave a report of the activities in his division during the year. State Veterinarian Robinson explained how his department had coöperated with federal veterinarians in fighting various diseases, such as bovine tuberculosis, Bang's disease and scabies.

A contribution of $25.00 to the A. V. M. A. Exhibit Fund was voted. Drs. Melody and Fauks were elected delegate and alternate, respectively, to the A. V. M. A. House of Representatives.

The Ladies' Auxiliary took an active part throughout the session and, at their luncheon on December 16, elected their officers for the ensuing year. Mrs. G. E. Melody was chosen president; Mrs. D. L. Cotton, secretary-treasurer, and Mrs. J. T. McGilvrary, chairman of the Committee on Local Arrangements.

G. E. MELODY, *Secretary.*

VERMONT VETERINARY MEDICAL ASSOCIATION

"Strictly Business" might well have been the motto for the annual meeting of the Vermont Veterinary Medical Association, held at Montpelier, December 10, 1938. No papers were presented, since the entire meeting was devoted strictly to matters of business.

Officers for the ensuing year were elected as follows: President, Dr. E. K. Treat, of Manchester; vice-president, Dr. J. J. Staab, of Montpelier; second vice-president, Dr. A. A. Mortimer, of Randolph, and secretary-treasurer, Dr. G. N. Welch, of Northfield.

A contribution of $10.00 was voted for the A. V. M. A. Exhibit Fund. Chief among the many matters decided at the session was the election of Dr. G. N. Welch as delegate to the A. V. M. A. House of Representatives. Dr. Welch will serve for the unexpired portion of the term of the late Dr. L. H. Adams and was granted the power to choose his own alternate.

G. N. WELCH, *Secretary.*

WESTERN NEW YORK VETERINARY MEDICAL ASSOCIATION

Fifty members attended the twenty-fifth annual meeting of the Western New York Veterinary Medical Association, held at Buffalo, December 15, 1938.

The session was opened with a clinic at the headquarters of the Erie County Society for the Prevention of Cruelty to Animals. Several cases were presented for examination and diagnosis, and an operation for the removal of a foreign body from the intestinal tract of a dog was performed by Drs. W. L. Weitz and R. B. McClelland, of Buffalo.

At the conclusion of the clinical program, a business session was held, Dr. J. V. Hills presiding. Officers for the coming year were elected as follows: President, Dr. Frank McBride, of Tonawanda; vice-president, Dr. G. D. Stone, of Ellicottville, and secretary-treasurer, Dr. F. F. Fehr (re-elected), of Buffalo.

Drs. R. A. McIntosh and F. H. Cote, of Guelph, Ont.; W. D. Rumney, of Hamilton, Ont.; J. M. Rice, of London, Ont., and E. B. Ingmand, of Indianapolis, Ind., were guests at the meeting.

In the evening, a banquet was held at the Hotel Touraine. Following the banquet, Dr. W. A. Dennis, of Jamestown, showed several reels of motion pictures of his travels in Europe, including the Thirteenth International Veterinary Congress, in Switzerland.

Dr. O. B. Webber, of Rochester, presented a paper on "Nonparasitic Skin Diseases of the Dog." Dr. Ingmand read a paper on "Equine Encephalomyelitis" and showed motion pictures of cases in various stages of the disease. Good discussions followed both speakers.

F. F. FEHR, *Secretary.*

INTERMOUNTAIN LIVESTOCK SANITARY ASSOCIATION

The Intermountain Livestock Sanitary Association held its twelfth annual meeting in Salt Lake City, Utah, January 9-11, 1939. A well-balanced program of scientific papers, informal group discussions, tours and parties filled out the most successful session in the history of this organization.

During the three-day period, the following papers were presented:

"Hypophosphatemia Associated with Parturient Hemoglobinemia of Dairy Cattle," Dr. D. E. Madsen, Logan, Utah.

"Equine Encephalomyelitis," Lt. Colonel R. A. Kelser, Washington, D. C.

"Some Factors Involved in Deficiency Diseases," Dr. A. H. Quin, Jr., Fort Dodge, Iowa.

"Recent Developments in the Study of Sheep Diseases," Dr. Hadleigh Marsh, Bozeman, Mont.

"Methods of Collecting and Evaluating Semen for Artificial Insemination of Sheep," Dr. C. E. Terrill, Dubois, Idaho.

"Observations on the Stiff-Lamb Problem," Dr. Edward Records, Reno, Nev.

"Recent Progress in Knowledge Concerning Tapeworms of the Genus *Moniezia*," Dr. W. H. Krull, Logan, Utah.

"Recent Investigations of Diseases Affecting Wild Life," Dr. E. R. Quortrup, Brigham, Utah.

"Food Allergy," Dr. Orin A. Ogilvie, Salt Lake City, Utah.

"The Army Horse Breeding Plan," Lt. Colonel J. W. Miner, Fort Douglas, Utah.

"Problems Relating to Municipal Milk Inspection," Mr. Ariel C. Merrill, Salt Lake City, Utah.

"The Keeping of Records in the Study of Problems Relating to Reproduction," Dr. H. E. Kingman, Cheyenne, Wyo.

"Bang's Disease: Past, Present and Future," Dr. Elmer Lash, Washington, D. C.

A resolution favoring the establishment of a federal regional laboratory for the eleven western states, the purpose of which would be to conduct research in animal dis-

eases as they occur under western range conditions, was passed by the Association.

Officers for the ensuing year were elected as follows: President, Dr. Hadleigh Marsh, of Bozeman, Mont.; first vice-president, Dr. J. C. Flint, of Salt Lake City, Utah; second vice-president, Dr. A. K. Kuttler, of Boise, Idaho; third vice-president, Dr. E. T. Powell, of Boise, Idaho, and secretary-treasurer, Dr. D. E. Madsen, of Logan, Utah.

D. E. MADSEN, *Secretary.*

INDIANA VETERINARY MEDICAL ASSOCIATION

Under the direction of its dynamic president, Dr. W. B. Massie, well-known figure of the veterinary service, the Indiana Veterinary Medical Association held its most successful meeting of recent years at the Severin Hotel, Indianapolis, January 10-12, 1939.

The attendance was exactly 300, of whom 185 were members, 62 were ladies, and the remainder visitors and guests. The program, which covered three full days, included a small-animal clinic and a banquet. Hon. George W. Gillie, past president of the association and now a congressman, addressed the banquet audience over a telephone hook-up from Washington, D. C. State Veterinarian Axby acted as toastmaster and Dr. L. A. Merillat, executive secretary of the national association, was the guest speaker.

The small-animal clinic, which has become one of the outstanding features of the Indiana association, was an excellent display of fine surgical work and was directed by Dr. F. R. Barlow. Dr. L. H. LaFond, of Detroit, Mich., was one of the principal operators. He was ably assisted by Drs. P. T. White, H. M. Hamilton, H. M. Bratt, R. J. Hoskins and H. W. Brown.

The literary program covered diseases of small animals, dairy practice, and diseases of swine and sheep. Equine encephalomyelitis and periodic ophthalmia were the best presented. The program consisted of:

"Nonparasitic Skin Diseases of the Dog; Contagious and Infectious Diseases of Cats," Dr. L. H. LaFond.

"The Veterinarian in His Relation to Public Health," Dr. F. W. Graves, of Albany, N. Y.

"Indiana Regulatory Work," Dr. J. L. Axby, of Indianapolis.

"Dairy Cattle Practice," Dr. S. L. Stewart, Olathe, Kan.

"Post-Vaccination Cases in Swine," Dr. L. W. Fox, Burt, Iowa.

"Indiana Swine Problems," Drs. T. L. Steenerson and G. M. Wagamon.

"Diseases of Sheep," Drs. L. D. Frederick and H. F. Emick.

Dr. L. P. Doyle, of Purdue University, analyzed the equine encephalomyelitis situation. He pointed out the close histopathology of the virus disease and that of toxic encephalomyelitis occurring in the fall and winter months. The perivascular cuffing, by which the disease is recognized microscopically, is practically identical in both cases, he explained. This paper was discussed by Drs. Walter F. Smith and O. C. Shockley.

Dr. W. W. Dimock, of the University of Kentucky, gave a fine address on his recent work on equine periodic ophthalmia, in which he brought out that, up to the present time, the researches under way on this disease under his direction have only succeeded in showing that little is known about the underlying etiological factors of the disease. Dr. T. A. Sigler, past president of the national association, well known for his wide experience in equine practice, discussed Dr. Dimock's address in detail. He emphasized the importance of this disease in horse production and felicitated the University of Kentucky on its undertaking. He pointed out that atropine sulfate, 3 per cent, instilled into the conjunctival sac, is the best means known to prevent given attacks from inflicting grave damage to the internal structures of the eyeball, but otherwise questioned the curative action of any other treatment known at the present time.

The following officers were elected: President, Dr. D. D. Baker, of Wabash; vice-president, Dr. R. E. Kepner, of Newcastle; members of the Executive Board, Drs. E. S. Hinkle, of Centerville, and J. L. Kixmiller, of Indianapolis, and secretary-treasurer, Dr. Charles C. Dobson (reëlected), of New Augusta.

The Executive Board discussed the important question of setting the dates of meetings of the associations of the adjacent states in such a way as to harmonize with the itinerary of commercial exhibitors and speakers.

OKLAHOMA VETERINARY MEDICAL ASSOCIATION

Over 100 veterinarians convened for the twenty-fourth annual meeting of the Oklahoma Veterinary Medical Association, held at the Skirvin Hotel, Oklahoma City, January 9-10, 1939.

Hon. Joseph C. Scott, president of the Oklahoma State Board of Agriculture, opened the session with an address of welcome, and the response was given by Dr. Walter H. Martin, of El Reno.

The following papers were presented:

"Modern Drugs in Modern Practice, and Modern Methods of Anesthesia," Dr. C. E. Fanslau, New York, N. Y.

"Canine Distemper Immunization and Canine Gastroenteritis," Dr. W. J. Purse, Kansas City, Mo.

"Poultry Practices," Dr. Lewis H. Moe, Stillwater.

"Medical and Surgical Equine Case Reports," Lt. Colonel J. A. McCallam, Fort Sill.

"Some Poisonous Plants of Oklahoma," Dr. H. I. Featherly, Stillwater.

"Artificial Insemination — Canine," Dr. W. F. Irwin, Tulsa.

"Equine Breeding Problems," Lt. Colonel R. T. Seymour, Fort Reno.

"Artificial Insemination in Large Animals," Dr. R. L. Anderes, Kansas City, Mo.

The officers for 1939 are: President, Dr. G. A. Cunningham, of Lawton; vice-president, Dr. C. H. McElroy, of Stillwater, and secretary-treasurer, Dr. F. Y. S. Moore (reëlected), of McAlester.

A highlight of the activities was the banquet and dance, held on the roof garden of the hotel.

F. Y. S. MOORE, *Secretary.*

VETERINARY MEDICAL ASSOCIATION OF NEW JERSEY

The fifty-fifth annual meeting of the Veterinary Medical Association of New Jersey was held at the Hotel Hildebrecht, Trenton, January 10-11, 1939, Dr. C. J. McAnulty presiding.

During this two-day session, the following papers were presented:

"Artificial Insemination," Dr. J. A. Henderson, Flemington, N. J.

"Medical and Surgical Diseases of the Eye," Dr. C. F. Schlotthauer, Rochester, Minn.

"Feline Enteritis," Dr. C. E. Salsbery, Kansas City, Mo.

"Nutrition and Herd Health," Dr. G. E. Taylor, New Brunswick, N. J.

"Equine Encephalomyelitis in New Jersey in 1938," Dr. R. A. Hendershott, Trenton, N. J.

"Hookworm Disease in Dogs," Dr. J. W. Landsberg, New York, N. Y.

"Some Everyday Problems in General Practice," Dr. W. H. Ivens, Philadelphia, Pa.

An outstanding feature of the meeting was the "dry" clinic. Two sessions were held, the purpose of which was to provide a series of brief, informal and practical presentations which would be of greater value and interest than the actual operative demonstrations.

Drs. J. B. Engle, of Summit; John Gray, of Newton; M. L. Morris, of Stelton; J. B. Hagenbuch, of Lawrenceville; R. P. Lawrence, of Verona; J. H. Spurlock, of Allentown; J. R. Varley, of New Brunswick; J. A. S. Millar, of Deal; P. F. Runyon, of Freehold; C. J. McAnulty, of Atlantic City, and C. F. Schlotthauer, of Rochester, Minn., were the clinical contributors.

An election of officers was held, with the following results: President, Dr. G. H. Kimnach, of Highstown; first vice-president, Dr. P. F. Runyon, of Freehold; second vice-president, Dr. John T. McGrann, of Trenton; treasurer, Dr. J. B. Engle, of Summit; secretary, Dr. J. G. Hardenbergh, of Plainsboro.

Among the guest speakers was Dr. William H. Ivens, of Philadelphia, Pa., first vice-president of the American Veterinary Medical Association, who discussed the publicity plans of the A. V. M. A. for 1939.

J. G. HARDENBERGH, *Secretary.*

DISTRICT OF COLUMBIA VETERINARY MEDICAL ASSOCIATION

The District of Columbia Veterinary Medical Association held its first quarterly meeting of the new year at the Mayflower Hotel, Washington, on January 17.

President Mason Weadon introduced Dr. George W. Gillie, Congressman from the Fourth District of Indiana, who discussed certain aspects of his recent campaign and election. Later, Dr. Gillie was made an honorary member of the Association during his term of office in Washington.

Dr. Adolph Eichhorn, of Beltsville, Md., spoke on "Pitfalls in Canine Distemper Prophylaxis." Dr. O. B. Hunter presented "Brucellosis in Man and Animals."

Dr. Hunter, a physician, was formerly professor of Veterinary Bacteriology and Pathology at George Washington University. In his talk he emphasized the remarkable results, both research and regulatory, which veterinarians have procured in the campaign to control Bang's disease. A significant fact brought out by the speaker is that *Brucella abortus* has been incriminated as a probable casual factor in Hodgkin's disease, according to laboratory studies made recently at Duke University but not yet published.

Officers for the ensuing year were elected as follows: President, Dr. H. W. Schoening; first vice-president, Lt. Colonel R. A. Kelser; second vice-president, Dr. Mason Weadon, and secretary-treasurer, Dr. Wm. M. Mohler.

WM. M. MOHLER, *Secretary.*

IOWA VETERINARY MEDICAL ASSOCIATION

The fifty-first annual meeting of the Iowa Veterinary Medical Association, held at Des Moines, January 17-19, 1939, appears to have broken all records for a "state" meeting. Among the 609 in attendance were 139 ladies. The rest were members, visitors and guests. The banquet, presided over by Dr. George R. Fowler, of Iowa State College, was attended by 639 and there were scores on the outside who could not be accommodated. The speakers were Hon. Geo. A. Wilson, Governor of Iowa; Hon. Mark G. Thornburg, Iowa Secretary of Agriculture; Dr. H. D. Bergman, president of the American Veterinary Medical Association, and Dr. R. G. Moore, president of the Iowa Veterinary Medical Association.

The commercial exhibits displayed adjacent to the meeting room were numerous and excellent additions to the glamor of the occasion. The technical program was interpolated throughout with lively arguments, indicating that the subjects and reporters were wisely chosen and the schedule well planned. It comprised:

"Equine Encephalomyelitis," Dr. L. R. Vawter, Reno, Nev.
"Report on Iowa Encephalomyelitis," Dr. H. A. Seidell, Des Moines.
"Remarks," Hon. Mark G. Thornburg, Des Moines.
"Veterinary Medical Organization," Dr. H. D. Bergman, Ames.
"Post-Vaccination Troubles," Dr. J. B. Winkel, Algona.
"Swine Erysipelas," Dr. J. A. Jensen, Marshalltown.
"Enteritis (of Swine)," Dr. C. G. Cole, Ames.
"Sheep Diseases," Dr. L. D. Frederick, Chicago, Ill.
"Bang's Disease," Dr. C. R. Donham, Ohio State University, Columbus, Ohio.
Idem, Dr. A. R. Menary, Cedar Rapids.
Idem, Dr. J. A. Barger, Des Moines.
"Undulant Fever," Dr. Carl F. Jordan, State Board of Health, Des Moines.
"Cattle Practice," Dr. D. E. Trump, Utica, Neb.
"Iodine," Dr. L. A. Merillat, Chicago, Ill.
"The Value of a Poultry Practice," Dr. P. V. Neuzil, Blairstown.
"Small-Animal Practice," Dr. J. C. Wright, Atlanta, Ga.
"Anesthesia in Small-Animal Practice," Dr. W. H. Riser, Des Moines.

The election leaves the official roster of the Association as follows: President, Dr. Frank Wilson, of Mechanicsville; president-elect, Dr. Guy M. Jones, of Audubon; first vice-president, Dr. W. A. Aitken, of Merrill; second vice-president, Dr. A. R. Menary, of Cedar Rapids, and secretary-treasurer, Dr. C. J. Scott, of Knoxville. Members of the Executive Committee: Drs. John Anstey, of Massena; Robt. D. Wall, of Des Moines; G. G. Baker, of Spencer; P. V. Neuzil, of Blairstown, and C. A. Stewart, of Tripoli.

Since Iowa maintains and faithfully supports a number of local associations, all of which meet at frequent intervals and attract good audiences, this large convention testifies to the fine development of organized veterinary medicine in that state. It seemed like a tribute to the president of the national association, whose labors can be pointed out as one of the factors which

consolidated the profession of agricultural Iowa and cultivated the spirit of coöperation so well expressed by this large and enthusiastic gathering.

A sorrowful event of the meeting was the sudden death of Dr. F. F. Parker (whose obituary appears elsewhere in this issue) at the dinner dance following the banquet. On this sad incident, Dr. J. A. Barger, acting for a special committee, read into the records of the meeting a solemn tribute to the memory of the departed member.

Foot-and-Mouth Disease in Sweden

In a letter just received from Professor H. Magnusson, Director of the Bacteriological Laboratory, Malmö, Sweden, some interesting facts concerning the actual struggles of his countrymen against the present severe outbreak of foot-and-mouth disease will undoubtedly be appreciated by many of our readers. Some details concerning the present outbreak and the results of emergency slaughter conducted until December 6 are as follows:

> Fourteen thousand, four hundred seventy-four cattle, 16,500 swine, and 736 sheep were slaughtered under an emergency decree. The indemnity payments amounted to eight million kronen (over two million dollars). The total number of infected premises from January, 1938, to December 6, 1938, were 1,787 for the entire country. Of this number, 1,636 were in the southern province of Schonen, where there is no slaughtering at present. The stamping-out procedure was carried on with equal intensity, especially in the most southern province, which is always exposed to infection from Denmark. There is a belief that the sparsely settled regions as well as those which are wooded are a better protection against the disease than the waters of the Sound between Denmark and south Sweden. Nevertheless, the infection has made great jumps and there are new cases occurring in central Sweden and even in Norway and Finland. The original cases could not be accounted for as the result of being carried by human individuals.
>
> In the most southern province of Schonen, where the emergency slaughtering method was stopped, there are now about 100 new cases daily. In Malmö and vicinity there are being used 3,000 liters of curative serum weekly.*
>
> The course of the disease has been variable. On many farms it seems to be benign; on others there is a mortality of 20 to 30 per cent, especially where there are numerous pregnant cows which abort in the acute stages. The Ministry of Public Health, about the middle of November, ordered the disinfection of all travelers from Denmark, but discontinued this procedure after three weeks because of public resistance. It was considered to be unreasonable by the people and was not carried out with sufficient thoroughness. At present, efforts are being confined to guarding the roads leading north from Schonen in order to prevent the transport of hogs and other hoofed animals northward from the infected province.

*I hope to receive the results of the use of this serum from Professor Magnusson in the near future. J. R. M.

From other sources it has been learned that, to many of the farmers, the isolation and segregation method will not be as welcome as slaughtering, because it is believed that the losses to the farmers during isolation will be greater.

It also has been reported in Sweden that, where the transportation of live stock is stopped by decree, the infection of foot-and-mouth disease is spread by feed-bags, automobiles, rats, dogs and birds, but the greatest spreader of the disease is man himself. The public has been warned to refrain from visiting the infected farms and to make telephone calls instead of personal calls wherever possible. On the Danish island nearest Sweden, over 20 per cent of the live stock is said to be infected. For this reason travelers arriving from this and other parts of Denmark are invited by the customs offices of Sweden to a room where they may wash their hands, face, hair and beard with a chloramine solution and then walk across a mat soaked with this disinfectant. In the meantime, the dust in their clothes is removed with the aid of a vacuum cleaner, and their shoes are polished. It was this disinfection order which raised a storm of protest and was discontinued after a trial for three weeks.

J. R. M.

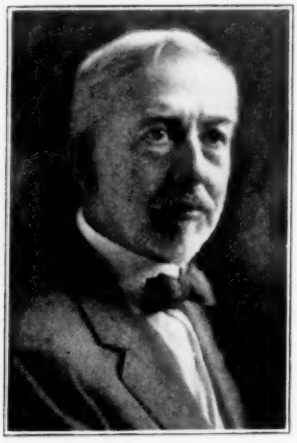
Of all the dispositions and habits which lead to political prosperity, religion and morality are indispensable supports.—*Washington.*

When friendship is settled, you must trust; before friendship is formed, you must pass judgment.—*Seneca.*

NECROLOGY

TAIT BUTLER

Dr. Tait Butler, of Memphis, Tenn., honor-roll member and past president of the A. V. M. A., died at his home on January 13, 1939. He had been in poor health for several months, but had been seriously ill only three weeks. Those who attended the Diamond Jubilee Convention of the A. V. M. A. in New York City the past summer will recall that Dr. Butler was in attendance and received one of the gold keys presented to the living past presidents.



DR. TAIT BUTLER

Born on a farm in Hastings County, Ontario, Canada, on July 24, 1862, Dr. Butler was educated at the Collegiate Institute, Saint Mary's, Ont. He then entered the Ontario Veterinary College and was graduated in 1885. He practiced in Iowa and Ohio for about six years and then accepted the appointment as professor of veterinary science and veterinarian to the experiment station at the institution which is now the Mississippi State College.

In 1895, Dr. Butler yielded to a desire to enter the publication field and founded the *Southern Farm Gazette*, at Starkville, Miss. He was editor and publisher for three years. Then he entered the service of the U. S. Bureau of Animal Industry and was stationed at Indianapolis, Ind., and Cudahy (Milwaukee), Wis. It was while he was a Hoosier that Dr. Butler was elected to the presidency of the A. V. M. A. He presided over the meeting held in Atlantic City in 1901.

Leaving government service at about this time, Dr. Butler went to Kansas as professor of veterinary science and bacteriology at Kansas State College. He was State Veterinarian of Kansas at the same time (1900-01). He then went to North Carolina as professor of veterinary science and zoölogy at the North Carolina Agricultural and Mechanical College (1901-06). He served also as State Veterinarian of North Carolina and as Director of Farmers' Institutes, until 1908.

In 1909, Dr. Butler went to Memphis, to become editor and vice-president of the *Progressive Farmer and Southern Ruralist*. From the start of this venture, he was an active crusader for farm abundance in the South. He emphasized the need for better farming methods and vigorously urged diversified agriculture through the breeding of more live stock and the developing of dairying. He was an ardent advocate of crop control and constantly preached the economy of feeding cottonseed meal to live stock.

Dr. Butler joined the A. V. M. A. in 1887 and was placed on the Honor Roll in 1937. Few members have served the organization in more ways. He was Resident Secretary for Mississippi (1891-92, 1895-97) and for North Carolina (1906-07). He was a member of the Executive Committee for six terms (1897-98, 1899-1901, 1902-03 and 1908-10) and served as chairman during three of these. He was a member of the

Committee on Diseases (1898-99), chairman of the Committee on Resolutions (1901-02) and member of the Committee on Standards of Excellence and Soundness (1903-04). He served on the Committee on Publications (1907-08), the Special Committee on Association of Faculties (1910-11), the Special Historical Committee (1913-15), the Committee on Reorganization (1915-16) and the Committee on Intelligence and Education (chairman, 1919-20).

In his presidential address, delivered at the 1901 convention, Dr. Butler gave many indications of his far-sighted vision. In commenting upon the progress made by the veterinary profession, he referred to it as "rather meteoric and brilliantly erratic," rather than "direct and conservative." In touching upon the veterinary educational system of the time, he pleaded for "more attention to the practical business side of veterinary education." He said further:

> There are too many teachers in veterinary colleges who never had any experience in general, unaided, independent veterinary practice of any sort, and too many others whose experience has been one-sided, although perhaps extensive.

Dr. Butler deplored our "blind, unreasoning worship of all things European," especially translations of German veterinary textbooks. He urged continued efforts to secure recognition for our Army veterinarians. He recommended the appointment of a committee to inaugurate a campaign to secure at least one competent veterinarian as a member of every board of health and every sanitary commission in the land. This was in 1901.

Other important posts held by Dr. Butler during his many-faceted career include: president of the Tennessee Cotton Growers Coöperative Marketing Association (1926-29); president of the American Association of Farmers' Institutes Workers (1937); director of the American Cotton Growers Exchange; secretary and later president of the Southern Cattlemen's Association; president of the Memphis Agricultural Club (1926-38); member of the Board of Governors of the Tennessee Hall of Fame.

In 1934, Dr. Butler was awarded a medal for distinguished service to agriculture by the American Farm Bureau Federation. He was one of the small number of veterinarians to win a place in "Who's Who."

Dr. Butler is survived by his widow (née Dell Bell), a son and a daughter.

LLOYD HERBERT MIDDAUGH

Dr. Lloyd H. Middaugh, of Memphis, Tenn., died at the Methodist Hospital, that city, on November 25, 1938, of peritonitis following an operation for appendicitis.

Born in Cameron, Mo., October 27, 1895, Dr. Middaugh attended local schools before entering the Kansas City Veterinary College. Following his graduation in 1918, he entered the service of the U. S. Bureau of Animal Industry. Since 1924, he had conducted the Small Animal Clinic in Memphis.

Dr. Middaugh joined the A. V. M. A. in 1919. He is survived by his widow (née Fay Green), his mother, two sisters and one brother.

J. H. G.

GEORGE M. LYNCH

Dr. George M. Lynch, of Sullivan, Wis., died suddenly on January 1, 1939, of a heart attack, while on a professional call.

Born at Elba, Wis., on November 30, 1885, Dr. Lynch was graduated from the Chicago Veterinary College in 1914. The year following his graduation, he practiced at Lomira and Hustisford, and located in Sullivan in 1916. For many years, he held the office of village assessor.

Dr. Lynch is survived by his widow (née Katharine Garity), four sons and one brother.

J. S. H.

VIVIAN W. YATES

Dr. Vivian W. Yates, of Memphis, Tenn., died at the Methodist Hospital, of that city, on January 3, 1939, of a heart ailment, from which he had suffered for over 20 years.

Born in Markle, Ind., May 22, 1897, Dr. Yates attended local schools, and then entered Chicago Veterinary College. Follow-

ing his graduation in 1918, he entered practice at Somerville, Tenn. From there, he moved to Clarksdale, Miss., and three years later to Ripley, Tenn., where he became very active in Baptist Church and Masonic circles. For the past two years, he had been in the service of the U. S. Bureau of Animal Industry and stationed in Memphis.

Dr. Yates joined the A.V.M.A. in 1936. He was a member of the Tennessee Veterinary Medical Association. He is survived by his widow (née Vivian Patmon), one son and two daughters. M. J.

FOSTER F. PARKER

Dr. F. F. Parker, of Des Moines,. Iowa, died suddenly following a heart attack, shortly after midnight, on January 19, 1939, while attending the annual ball of the Iowa Veterinary Medical Association. He had suffered a slight heart attack three years

DR. F. F. PARKER

earlier, from which he appeared to have recovered.

Born at Lowden, Iowa, February 22, 1875, Dr. Parker was graduated from the Wall Lake (Iowa) High School and then entered the Iowa State College for the study of veterinary medicine. He was graduated in 1900 and entered practice at Oskaloosa, Iowa. At the same time, he was head coach of the Penn College football team of that city. Dr. Parker had been left halfback on the first Cyclone team in 1898, and his outstanding ability as a player had resulted in his selection as head football coach during his senior year at Ames.

Later, Dr. Parker became chief veterinarian of the National Live Stock Insurance Company, but he resigned this position to enter small-animal practice in Des Moines, where he operated a hospital for a number of years.

Dr. Parker joined the A. V. M. A. in 1924. He served as chairman of the Section on Small Animals (1930-31) and as member of the A. V. M. A. Executive Board for District 5 (Iowa and Minnesota) for one year (1934-35). He had been a member of the Iowa Veterinary Medical Association since 1900 and, at one time, served as president of the organization. He was a member of the Twelfth International Veterinary Congress and the Conopus Club, a Scottish Rite Mason, and a Shriner.

His mission in life seemed to be to gain knowledge in his profession through careful study and association with those who were pledged to lives of usefulness, and to pass this knowledge on to those who were ever appreciative of a character and personality such as his.

The survivors are his widow, one daughter, and two sons. J. A. B.

W. B. NILES

Dr. W. B. Niles, known in scientific circles all over the world for his work in connection with hog cholera, died at his home in Covina, Calif., on January 23, 1939. He had been in poor health for several years and underwent an operation about a week before his death.

Born in Rock County, Wis., on November 5, 1855, of New England ancestry, Dr. Niles was reared on a farm and received his early education in rural schools. When he was 15, the family moved to Iowa. In the spring of 1884, he entered Iowa State College for the study of veterinary medicine. The following year he was graduated as an honor student. In 1886, he was appointed House Surgeon at the Veterinary School, working under the direction of Dr.

Milliken Stalker, who was also State Veterinarian of Iowa. In the fall of 1886, Dr. Niles was appointed State Veterinarian.

Shortly after this appointment, Dr. Niles decided to enter private practice and located at Webster City, Iowa, where he remained until July, 1887, when he accepted the appointment of professor of veterinary science and veterinarian to the State Agricultural Experiment Station at the University of South Carolina, at Columbia. He remained in this position until 1890.

In 1891, Dr. Niles returned to Iowa State College. He taught various subjects, including surgery, obstetrics and materia medica, and had charge of the veterinary clinic. As assistant veterinarian at the Iowa Agricultural Experiment Station, he conducted research work on glanders, actinomycosis, bovine tuberculosis and hog cholera.

Dr. Niles entered the service of the U. S. Bureau of Animal Industry in 1898, and for two years was in charge of hog cholera investigations in Fremont County, Iowa. In 1900, he was transferred to meat inspection work in Omaha, but a short time later was sent to Sidney, Iowa, to take charge of experimental work in connection with hog cholera. In 1905, the work was transferred to Ames.

During the two years immediately following, Dr. Niles did his epochal work with anti-hog cholera serum. This was started under the direction of Dr. E. A. de Schweinitz and continued under the direction of Dr. M. Dorset, late chief of the Biochemic Division of the U. S. Bureau of Animal Industry. Dr. C. N. McBryde was associated in this work and the three names—Dorset, Niles and McBryde—will always be identified with anti-hog cholera serum, the most extensively used biological product in the world.

Dr. Niles joined the A. V. M. A. in 1891. He was Resident Secretary for Iowa (1892-93), member of the Committee on Intelligence and Education (1893-94, 1896-98), member of the Committee on Diseases (1895-96), member of the Committee on Publications (1897-98), member of the Committee on Resolutions (1899-1900) and member of the Special Committee on Revision of Constitution and By-Laws (1910-11).

In 1928, Dr. Niles retired from active service and, two years later, removed to California. While a resident of Ames, he was prominently identified with Boy Scout work. Surviving him are his widow, two sons, two daughters, one brother and two sisters.

WILLIAM S. LONGACRE

Dr. William S. Longacre, of Lehighton, Pa., died on January 4, 1939, as the result of septicemia which developed following a nail puncture of one hand on December 17, 1938.

Born in West Penn Township, Carbon County, Pa., on August 19, 1872, Dr. Longacre attended the Keystone State Normal School and the Ontario Veterinary College. Following his graduation from the latter institution in 1896, he entered practice at Lehighton.

Dr. Longacre joined the A. V. M. A. in 1927.

SAMUEL M. SPRINGER

Dr. Samuel M. Springer, of Indianapolis, Ind., aged 77 years, died at his home on January 12, 1939, after a long illness. He was a graduate of the Indiana Veterinary College, class of 1898. He later took postgraduate work at the same institution and received a degree again in 1904. He was in the service of the U. S. Bureau of Animal Industry for about 20 years. He is survived by his widow, one daughter and two sons.

Our sympathy goes out to Dr. B. H. Steigleder (Cin. '16), of Chicago, Ill., in the death of his wife (née Eda Bell Starn), on January 3, 1939.

Modesty is to the other virtues in a man, what shade in a picture is to the parts of the thing represented. It makes all the other beauties conspicuous, which would otherwise be but a wild heap of colors.—*Sir R. Steele.*

GLEANINGS FROM OUR CORRESPONDENCE

BIRTHS

To Dr. and Mrs. J. J. Arnold, of Newcastle, Ind., a son, Charles Allen, September 12, 1938.

To Dr. and Mrs. Nelson Howe, of Saint Johns, Mich., a daughter, October 20, 1938.

To Dr. and Mrs. E. E. Rash, of Iowa Falls, Iowa, a son, November 5, 1938.

To Dr. and Mrs. E. H. Gloss, of Gaylord, Minn., a son, Robert Cole, December 1, 1938.

To Dr. and Mrs. J. R. Karr, of Coshocton, Ohio, a son, Richard Duncan, January 12, 1939.

MARRIAGES

Dr. Leonard Wilbur Hibbs (K. S. C. '35), of Upland, Calif., to Miss Viola Hegness, of Alcester, S. D., at Alcester, December 30, 1938.

Dr. Edward S. White (Mich. '38), of Richmond, Va., to Miss Elizabeth Jackson, of Holt, Mich., at Richmond, January 1, 1939.

PERSONALS

Dr. L. K. Green (U. P. '94) is mayor of Auburn, Me.

Dr. A. W. Winter (Mich. '38) has established a practice at 205 E. Dewey Ave., Buchanan, Mich.

Dr. H. C. Rhodes (U. P. '16), of Saint Albans, Vt., is taking an extended vacation in Florida.

Dr. Clayton Stephens (Ala. '38) is now associated with Dr. J. T. Alston (Chi. '11), at Tupelo, Miss.

Dr. E. N. Kestner (Cin. '11), of Brownstown, Ind., has been appointed Chief Deputy State Treasurer of Indiana.

Dr. George R. Moore (Mich. '38) is teaching pharmacology and therapeutics at Kansas State College, Manhattan, Kan.

Dr. E. E. Slatter (O. S. U. '30), of Danville, Ill., addressed the Vermilion County (Ill.) Rural Youth Group recently on "Rabies."

Dr. H. E. Ryan (K. S. C. '36), formerly of Marshfield, Wis., is now engaged in general practice at Vassar, Mich.

Dr. Silas Gaynor (Ind. '20), of Logansport, Ind., recently addressed the American Legion Service Club on "Modern Veterinary Science."

Dr. John Gwaltney (Ind. '15) has resigned from the service of the U. S. Bureau of Animal Industry to enter private practice at Centralia, Ill.

Dr. J. W. Cunkleman (Mich. '38), of Perry, Mich., has been appointed instructor in anatomy at Michigan State College, East Lansing, Mich.

Dr. John W. Jackman (O. S. U. '19), of Columbus, Ohio, has been reappointed a member of the Ohio State Board of Veterinary Examiners.

Dr. Otto Augspurger (St. Jos. '08), of Chicago, Ill., was retired from the service of the U. S. Bureau of Animal Industry, on December 31, 1938.

Dr. James D. Nundy (Mich. '38), of Bellaire, Mich., has been appointed County Veterinarian by the Antrim County (Mich.) Veterinary Service Association.

Dr. Ray Bankowski (Mich. '38) is now engaged in poultry disease research under Dr. J. R. Beach (Corn. '13), at the University of California, Berkeley.

Dr. F. J. Bardsley (U. S. C. V. S. '15), of Burlington, Vt., who has been incapacitated on account of coronary thrombosis, is reported to be somewhat improved.

Dr. W. P. McKenzie (Ont. '38), who formerly was associated with Dr. A. J. Neal, at Bangor, Me., is now with Dr. A. E. Cleaves, of Franklin Roads, Me.

Dr. Charles R. Corson (Mich. '38), of Elkhart, Ind., has taken over the practice of the late Dr. P. E. Schwin and is building a new small-animal hospital.

Dr. E. G. Sadler (Corn. '32), of Ellsworth, Me., who formerly was associated with Dr. A. E. Cleaves, of Franklin Roads, Me., has been employed by the Thirlstane Ranch, Bar Harbor, Me.

Dr. F. E. Myers (Ont. '12), of Gladwin, Mich., has been appointed to the Michigan State Veterinary Examining Board. He will replace Dr. John A. Schaefer, who moved from the state last July.

Dr. S. E. Douglas (West. '06), of Oklahoma City, Okla., was struck by an automobile recently while crossing the street in front of his home, and suffered a very severely bruised ankle and knee.

Dr. H. W. Ayers (Ont. '08), of Oklahoma City, Okla., is suffering from a broken hip, as the result of a fall in his office, on December 17, 1938, and has been confined in the hospital since that date.

Dr. A. G. Gieske (Chi. '06) has built the Barrington Animal Hospital, 216 S. Northwest Highway, Barrington, Ill., and is now operating this unit for small animals in conjunction with his general practice.

Dr. George Fohey (Mich. '38), of Clio, Mich., who was guest speaker at a recent meeting of the Clio Rotary Club, talked on modern methods in animal surgery, pointing out the progress that has been made by the veterinary profession during the past ten years.

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